
ASSESSMENT REPORT ON

PENTACHLOROPHENOL

FOR DEVELOPING

AMBIENT AIR QUALITY

OBJECTIVES



**ASSESSMENT REPORT ON
PENTACHLOROPHENOL
FOR DEVELOPING AMBIENT AIR QUALITY OBJECTIVES**

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FOREWORD

Alberta Environment maintains Ambient Air Quality Objectives¹ to support air quality management in Alberta. Alberta Environment currently has ambient objectives for thirty-one substances and five related parameters. These objectives are periodically updated and new objectives are developed as required.

With the assistance of the Clean Air Strategic Alliance, a multi-stakeholder workshop was held in October 2000 to set Alberta's priorities for the next three years. Based on those recommendations and the internally identified priority items by Alberta Environment, a three-year work plan ending March 31, 2004 was developed to review four existing objectives, create three new objectives for three families of substances, and adopt six new objectives from other jurisdictions.

This document is one of a series of documents that presents the scientific assessment for these adopt substances.

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Science and Standards Branch

¹ **NOTE:** The *Environmental Protection and Enhancement Act*, Part 1, Section 14(1) refers to "ambient environmental quality objectives" and uses the term "guidelines" in Section 14(4) to refer to "procedures, practices and methods for monitoring, analysis and predictive assessment." For consistency with the *Act*, the historical term "ambient air quality guidelines" is being replaced by the term "ambient air quality objectives." This document was prepared as the change in usage was taking place. Consequently any occurrences of "air quality guideline" in an Alberta context should be read as "air quality objective."

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SUMMARY

The main objective of this assessment report was to provide a review of scientific and technical information to assist in evaluating the basis and background for an ambient air quality guideline for pentachlorophenol.

Pentachlorophenol is a white, needle-like crystal solid that does not occur naturally. Pentachlorophenol is no longer manufactured in Canada, but it is imported for use primarily as a wood preservative. It is also used as a pesticide, herbicide, insecticide, fungicide, biocide, bactericide, and algacide, and as additives to other products such as adhesives and drilling muds. Pentachlorophenol is not listed as a National Pollutant Release Inventory (NPRI) substance. Emissions of pentachlorophenol to the atmosphere result from its transportation, storage and use.

With regard to acute (short-term) pentachlorophenol exposure in humans, irritation of eyes, nose, and throat was reported at concentrations greater than 1 mg/m^3 (0.09 ppm) based on exposures reported as an 8-hour time weighted average. A rise in metabolic rate, hypothermia and effects on the central nervous system, respiratory system, cardiovascular system, liver and blood has also been reported; however, at exposure concentrations not specified. Studies on infants and children suggest a greater susceptibility of children to acute pentachlorophenol toxicity. Acute and sub-acute inhalation exposure of animals to pentachlorophenol has been reported to affect the respiratory system, blood, liver, kidney and adrenal system. Most of these effects have been observed based on exposure concentrations ranging from 3 to 21 mg/m^3 (0.3 to 1.9 ppm) in studies where exposures occurred four hours/day, six days/week over four months.

Accidental exposure to pentachlorophenol in the workplace represents situations where acute (short-term) exposure conditions could occur. However, these conditions are addressed through occupational health and safety programs. With respect to non-occupational settings, it is unlikely that acute exposures to pentachlorophenol in these settings would be persistent enough to be a public health concern in Alberta. Most of the agencies reviewed have short-term (1-hour and/or 24-hour) air quality guidelines for pentachlorophenol for acute exposure conditions. Six US state agencies adopted short-term guidelines for pentachlorophenol from occupational exposure limits. One-hour guidelines range from 0.026 to $60 \text{ } \mu\text{g/m}^3$ among the various agencies, while 24-hour guidelines range from 0.003 to $100 \text{ } \mu\text{g/m}^3$ among the agencies.

In human occupational studies, chronic pentachlorophenol inhalation has been reported to be associated with effects on the respiratory system, endocrine system, central nervous system, and reproductive system. Adverse effects on liver and blood were also reported. However, these occupational studies carried a high potential for confounding exposure factors that limits their interpretation. Consequently, exposure concentrations relating to the reported effects are uncertain. No chronic inhalation animal studies were identified. Chronic animal ingestion studies reported effects on the liver and kidney; endocrine and reproductive system effects; developmental effects, and carcinogenicity.

Most of the agencies whose air quality guidelines were reviewed have chronic (long-term) guidelines for pentachlorophenol. The states of California, Michigan, and New Jersey use a carcinogenic endpoint to derive their respective guideline – using the US EPA's oral unit risk of 0.12 per mg/kg/day. Several of the agencies also have a chronic guideline for non-carcinogenic effects – using occupational exposure limits (New Hampshire, Texas, and Vermont). Annual average guidelines range from 0.029 to 2 $\mu\text{g}/\text{m}^3$ among the various agencies.

1.0 INTRODUCTION

Alberta Environment establishes Ambient Air Quality Objectives under Section 14 of the *Environmental Protection and Enhancement Act* (EPEA). These objectives are part of the Alberta air quality management system (AENV, 2000).

The main objective of this assessment report was to provide a review of scientific and technical information to assist in evaluating the basis and background for an ambient air quality objective for pentachlorophenol. The following aspects were examined as part of the review:

- physical and chemical properties,
- existing and potential anthropogenic emissions sources in Alberta,
- effects on humans, animals, and vegetation,
- ambient air guidelines in other Canadian jurisdictions, United States, World Health Organization and New Zealand, and the basis for development and use,
- monitoring techniques

Important physical and chemical properties that govern the behaviour of pentachlorophenol in the environment include, but are not limited to, chemical structure, molecular weight, melting and boiling points, water solubility, density, vapor density, organic carbon partition coefficient, octanol water partition coefficient, vapor pressure, Henry's Law constant, bioconcentration factor, and odor threshold. Values for these properties were reviewed and presented in this report.

Existing and potential natural and anthropogenic sources of pentachlorophenol emissions in Alberta was examined. Environment Canada's National Pollutant Release Inventory (NPRI) did not list anthropogenic emissions for pentachlorophenol in 2001.

Scientific information about the effects of pentachlorophenol on humans and animals is reported in published literature and other sources. Studies were primarily identified from peer reviewed evaluations on humans and animals undertaken by the Agency for Toxic Substances and Disease Registry (ATSDR, 2001) and the World Health Organization (WHO, 1987), and from studies listed in the Hazardous Substances Databank (HSDB, 2002). These sources provided valuable information for understanding health effects of pentachlorophenol exposure.

Ambient air guidelines for pentachlorophenol are used by a number of jurisdictions in North America for different averaging-time periods. These guidelines can be developed by using an occupational exposure level and dividing it by safety or adjustment factors, using non-cancer risk assessment procedures, or by using cancer risk assessment procedures. The basis for how these approaches are used by different jurisdiction to develop guidelines was investigated in this report.

Air sampling and analytical methods for pentachlorophenol used in practice by regulatory agencies were reported. In general, standard air monitoring methods for pentachlorophenol are based on high and low volume solid sorbent or pump-and-tube sampling approaches. Widely employed and accepted reference air monitoring methods for pentachlorophenol have been developed, tested and reported by the United States Environmental Protection Agency (US EPA), National Institute of Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA). These methods were reviewed and presented in this report.

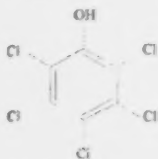
2.0 GENERAL SUBSTANCE INFORMATION

Pentachlorophenol is a white, needle-like crystal solid (IPCS, 1987) with a phenolic odour (Genium, 1999). Pentachlorophenol is stable but incompatible with strong oxidizers and alkalies (Genium, 1999). Decomposition of pentachlorophenol may lead to the formation of hydrogen chloride, chlorine or chlorinated hydrocarbons (Genium, 1999).

Pentachlorophenol is no longer produced in Canada but it is; however, imported for use (Health Canada, 1987). Pentachlorophenol is used primarily as a wood preservative (ATSDR, 2001). Use as a wood preservative accounts for the majority of the chlorophenol use in Canada (Health Canada, 1987). Other uses of pentachlorophenol include use in pesticides, herbicides, insecticides, fungicides, biocides, bactericides, algaecides and anti-mildew agents; as additives to adhesives; in shingles, roof tiles, and other products such as drilling muds in the petroleum industry (Genium, 1999).

Table 1 provides a list of important identification numbers and common synonyms for pentachlorophenol.

Table 1 Identification of Pentachlorophenol (Genium, 1999)

Property	Value
Formula	C_6HCl_5O
Structure	
CAS Registry number	87-86-5
RTECS number	SM6300000
UN Number	UN2671, UN2762, UN2995, NA2020
Common Synonyms/Tradenames	Acutox; Chem-Penta; Chlon; Chlorophen; Cryptogil Oil; Cryptogil OL; Dow Pentachlorophenol DP-2 Antimicrobial; Dowcide 7; Dowcide 6; Dowcide 7; Dowcide 7 Antimicrobial; Dowcide EC-7; Dura Treet II; Durotox; EP 30; EPA Pesticide Chemical Code 063001; Forpen-50 Wood Preservative; Fungifen; Glazd Penta; Grundier Arbezol; 1-hydroxypentachlorobenzene; Lauxtol; Lauxtol A; Lioprem; Ontrack We Herbicide; Ortho Triox Liquid Vegetation Killer; Osmose Wood Preserving Compound; PCP; Penchlorol; Penta; Penta Concentrate; Penta Ready; Penta WR; Pentachlorophenate; 2,3,4,5-pentachlorophenol; Pentachlorophenol, Dowcide EC-7; Pentachlorophenol, DP-2; Pentachloropheno, Technical; Pentachlorophenol; Pentacon; Penta-kil; Pentasol; Penwar; Peratox; Permicide; Permagard; Permasan; Permatox DP-2; Permatox Penta; Permite; Prevenol; Priltox; Santobrite; Santophen; Santophen 20; Sinithuo; Term-I-Trol; Thompson's Wood Fix; Watershed Wood Preservative; Weed and Brush Killer; Weedone; Witophen P; Woodtreat; Woodtreat A

2.1 Physical, Chemical and Biological Properties

The physical and chemical properties of pentachlorophenol are summarized in Table 2.

Table 2 Physical and Chemical Properties of Pentachlorophenol

Property	Value	Reference
Molecular Weight	266.34	Lide, 2002
Physical state	White monoclinic crystalline solid Technical grade dark gray to brown	Verschueren, 2001
Melting Point	174 °C	Lide, 2002
Boiling Point	310 °C (decomposes)	Lide, 2002
Specific gravity (liquid)	1.978 (at 22°C)	Lide, 2002
Specific gravity (gas) (air =1)	9.20	Verschueren, 2001
Vapour pressure	0.00011 mm of Hg (at 20°C)	Verschueren, 2001; Howard, 1991
Solubility in water	Slightly soluble in water	Lide, 2002
	0.13% (% weight at 25°C)	Lide, 2002
	5 mg/L at 0°C	
	14 mg/L at 20°C	Verschueren, 2001
	35 mg/L at 50°C	
Solubility	Soluble in benzene, very soluble in ethanol and ether	Lide, 2002
PKa	4.60 – 5.30	Mackay <i>et al.</i> , 1992
Henry's Law Constant	$2.75 \times 10^{-6} \text{ atm} \cdot \text{m}^3 \cdot \text{mol}^{-1}$	Howard, 1991
	0.0248 to 0.284 $\text{Pa} \cdot \text{m}^3 \cdot \text{mol}^{-1}$	Mackay <i>et al.</i> , 1992
Octanol water partition coefficient (log K_{ow})	5.07	Lide, 2002
	4.07; 5.01	Verschueren, 2001
Organic carbon partition coefficient (K_{oc})	293 to 900 (at 0.0125 mg/L)	Verschueren, 2001
	1000 (calculated)	Howard, 1991
	3000 to 4000 (measured)	Howard, 1991
Flash Point	Not flammable	Weiss, 1986
Explosive limits	No data	ATSDR, 2001
Autoignition temperature	Not flammable	Weiss, 1986
Odour threshold	0.857 mg/L at 30°C (in water)	Verschueren, Hoak cited in ATSDR, 2001
	12.0 mg/L at 60°C (in water)	
Bioconcentration factor in fish (log BCF)	-2.66 to 8.00	Mackay <i>et al.</i> , 1992
	Depends on pH	
	0.3 to 5.5	Howard, 1991
Conversion factors for vapour (at 25 °C and 101.3 kPa)	1 $\text{mg}/\text{m}^3 = 0.09 \text{ ppm}$ 1 $\text{ppm} = 11.1 \text{ mg}/\text{m}^3$	Verschueren, 2001

2.2 Environmental Fate

The environmental fate of pentachlorophenol is summarized in Table 3. Pentachlorophenol, once released to the atmosphere, may be removed via wet and dry deposition or via reactions such as photolysis or oxidation (ATSDR, 2001). Pentachlorophenol released to soil will adsorb/absorb to particles and biodegrade while pentachlorophenol released to water will undergo photolysis and biodegradation (ATSDR, 2001).

Table 3 Environmental Fate of Pentachlorophenol (Howard, 1991)

System	Fate	Reaction Rates
Water	• will dissociate • dissociated form will photodegrade • may biodegrade • will adsorb to sediment • hydrolysis and volatilization are negligible	• <i>photodegradation</i>: half life of hours to days
Soil	• partitions mainly to soil • hydrolysis and photolysis are negligible • will biodegrade • if not dissociated, will volatilize • if dissociated, leaching to groundwater is possible • may be associated with particulate matter and settle out by gravitational settling	• <i>biodegradation</i>: half life of weeks to months
Air	• in the vapour phase, will be subject to photolysis and potentially photochemical reactions with hydroxyl radicals	• <i>photolysis</i>: 6.2%/hour • <i>photochemical reactions</i>: 1.5%/hour

3.0 EMISSION SOURCES AND INVENTORIES

3.1 Natural Sources

Production of pentachlorophenol during fungus metabolism has been suggested as a possible natural source of pentachlorophenol (IARC cited in Howard, 1991); however, according to ATSDR (2001), pentachlorophenol is a synthetic substance and does not occur naturally.

3.2 Anthropogenic Sources

3.2.1 Industrial Sources

Pentachlorophenol may be released to the atmosphere during its production, however this release is considered negligible since there are few facilities currently producing pentachlorophenol (ATSDR, 2001). In the United States, pentachlorophenol is only produced by Vulcan Materials (in Wichita, Kansas) (ATSDR, 2001). In Canada; however, pentachlorophenol is no longer manufactured (Health Canada, 1987) and/ therefore, there are no emissions from production facilities.

Pentachlorophenol was not a National Pollutant Release Inventory (NPRI) substance in 2001.

3.2.2 Other Sources

Pentachlorophenol may also be released to the atmosphere during its storage, transportation, or use, mainly as a wood preservative (Howard, 1991). Pentachlorophenol may volatilize during the wood treating process, after accidental spills or directly from the treated wood products (Howard, 1991). Use of pentachlorophenol as a fungicide, herbicide, bactericide, algacide or its use in leathers and textiles or in the manufacturing of other products, may all contribute to pentachlorophenol emissions (Howard, 1991). In the past, the use of pentachlorophenol as an anti-fouling agent in cooling towers also contributed to pentachlorophenol emissions (ATSDR, 2001). Incineration of chlorine-containing wastes may also be an anthropogenic source of pentachlorophenol to the atmosphere (ATSDR, 2001).

3.3 Ambient Levels

Cessna *et al.* (1997) measured ambient levels of pentachlorophenol in the vicinity of select Canadian cities (Regina, Waskesiu and Yellowknife). Pentachlorophenol concentrations ranged from 0.06 to 3.68 ng/m³. As part of the Detroit Incinerator Monitoring Program, Environment Canada (cited in Cessna *et al.*, 1997) reported a mean ambient air pentachlorophenol concentration of 0.63 ng/m³ for two sites in southern Ontario. Kumar (2001), in a study of pesticides in ambient air in Alberta, measured pentachlorophenol concentrations ranging from 0 to approximately 2.8 ng/m³ at four locations in Alberta (Lundbreck, Lethbridge, Lacombe and Vegreville). The study identified pentachlorophenol as a pesticide of interest in ambient air.

4.0 EFFECTS ON HUMANS, ANIMALS, AND VEGETATION

The purpose of this assessment is to provide a summary and interpretation of the available toxicological and epidemiological studies on the health effects following the inhalation of pentachlorophenol (PCP). Studies were primarily identified from peer-reviewed evaluations on humans and animals undertaken by the Agency for Toxic Substances and Disease Registry (ATSDR, 2001) and the World Health Organization (WHO, 1987), and from studies listed in the Hazardous Substances Databank (HSDB, 2002). The focus of this assessment was adverse health effects associated with inhalation exposure to pentachlorophenol. Due to a lack of chronic inhalation studies in animals and considering the mechanism of action for pentachlorophenol, oral-based studies were also identified for exposure of animals to pentachlorophenol. Two types of effects – acute and chronic – were examined. Acute effects usually occur rapidly as a result of short-term exposures, and are of short duration (generally for exposures less than 24 hours; Gallo, 1996). Chronic effects generally occur as a result of long-term exposure, and are of long duration (generally as repeated exposures for more than 12 months; Gallo, 1996).

Exposure-response data from key toxicological and epidemiological studies were summarized in table form to provide a quick reference to health effects observed in critical receptors over a defined period of inhalation exposure to pentachlorophenol. The relevance of this data to public health is then discussed.

4.1 Overview of Chemical Disposition

The non-polar, lipophilic properties of pentachlorophenol enable ready absorption through cell membranes of the lungs, gastrointestinal tract and skin (ATSDR, 2001). Human and animal studies reported at least 75% absorption following short-term inhalation exposure (ambient concentrations of 0.230 and 0.432 ng/m³ (Casarett *et al.* cited in ATSDR, 2001; Hoben *et al.* cited in WHO, 1987).

The distribution of PCP is influenced by binding to plasma proteins (estimated to be as high as 95%) (Braun *et al.* cited in ATSDR, 2001). Protein binding increases with increasing inhalation dose (Hoben *et al.* cited in WHO, 1987). In humans, absorbed pentachlorophenol is distributed to the adipose tissue, liver, lungs, kidneys, blood, brain and spleen (Shafik cited in ATSDR, 2001; Ohe cited in WHO, 1987; Grimm *et al.* cited in ATSDR, 2001). Pentachlorophenol has also been detected at low concentrations in breast milk (Gebefugi and Korte cited in WHO, 1987; Veningerova *et al.* cited in ATSDR, 2001). A number of chlorinated contaminants (hexachlorobenzene, hexachlorocyclohexane, pentachloronitrobenzene and lindane) are metabolized to pentachlorophenol; therefore, the detection of pentachlorophenol at these sites may not be directly related to pentachlorophenol exposure (WHO, 1987; ATSDR, 2001). In rats, pentachlorophenol was distributed to the liver, lungs and blood following acute inhalation exposure (Hoben *et al.* cited in WHO, 1987).

Metabolism of PCP occurs in the liver, the major pathways are conjugation (glucuronide formation) and oxidative dechlorination (tetrachlorohydroquinone formation). In human and animals, the majority of absorbed pentachlorophenol is excreted in the urine unchanged; metabolism is likely prevented due to binding with plasma proteins. Differences in elimination

were noted for different species, sex, route and duration of exposure (Hoben *et al.* cited in WHO, 1987; Braun *et al.*, Reigner *et al.*, Braun and Sauerhoff, Braun *et al.* cited in ATSDR, 2001).

A urinary excretion half-life of 33 hours was reported in humans following a single oral dose (0.1 mg/kg) of pentachlorophenol; the authors reported that urinary excretion followed first-order kinetics (Braun *et al.*, 1979). However, Uhl *et al.*, (cited in Fisher, 1991) reported a much longer elimination half-life (17 days) based on cases of human pentachlorophenol poisonings. A comparison of urine and blood concentrations in individuals with non-occupational or no known exposure to PCP indicated that PCP levels in serum or plasma were generally higher than PCP levels in urine. This was in contrast to cases of lethal intoxication, where PCP levels were higher in urine than blood (WHO, 1987).

As with many toxicants, as the body burden lowers, the pattern of elimination for PCP is more complex (i.e., does not follow first-order kinetics) and accumulation can occur over a chronic exposure period if absorption rates exceeds elimination rate (ATSDR, 2001). There is limited evidence for long-term accumulation and storage of pentachlorophenol, with higher concentrations detected in the liver and kidney, and lower concentrations in body fat, brain and muscle tissue (Uhl *et al.* cited in Fisher, 1991; WHO, 1987).

There is evidence that PCP is most toxic through the inhalation route of exposure; however, similar effects in humans and animals via all routes and duration of exposure demonstrate that PCP is highly toxic, regardless of the route, length, and frequency of exposure WHO (1987). The toxic effects reported in humans and animals following pentachlorophenol exposure are likely due to the increase in cellular aerobic metabolism and increased heat production (hyperthermia) caused by the uncoupling of mitochondrial oxidative phosphorylation (WHO, 1987; ATSDR, 2001). The evidence in rats of binding of pentachlorophenol to liver mitochondrial protein supports the potential for alteration of the enzymes involved in oxidative phosphorylation (Weinbach and Garbus cited in WHO, 1987; ATSDR, 2001).

4.2 Genotoxicity

Genotoxicity studies performed on blood collected from workers occupationally exposed by inhalation to pentachlorophenol or its sodium salt (exposure concentrations unknown) reported no sister chromatid exchange in isolated lymphocytes (Bauchinger *et al.* cited in HSDB, 2002; Wyllie *et al.* cited in WHO, 1987; Ziemson *et al.* cited in HSDB, 2002). Two of these studies also found no chromosomal aberrations (Wyllie *et al.* cited in WHO, 1987; Ziemson *et al.* cited in HSDB, 2002). Data on exposure duration (3 to 34 years), ambient air concentrations of pentachlorophenol (1.2 to 180 $\mu\text{g}/\text{m}^3$) and blood plasma pentachlorophenol concentrations in healthy workers (23 to 775 $\mu\text{g}/\text{L}$) were reported for one study where no effects on chromosomal aberrations or sister chromatid exchange were observed (Ziemsens, *et al.* cited in HSDB, 2002).

The results of genotoxicity studies of mice exposed *in vivo* to pentachlorophenol (exposure concentrations not reported) were mixed. In general, an increase in DNA adduct formation in liver cells was reported following oral exposure to mice (Sai-Kato *et al.*, Umemura *et al.* cited in ATSDR, 2001) whereas, no evidence of genotoxicity (micronuclei formation, gene mutations

and recombination) was detected in a mouse spot test assay and following intraperitoneal exposure to mice and rats (NTP, 1999; Fahrig and Steinkamp-Zucht cited in ATSDR, 2001).

No evidence of genotoxicity was determined by other *in vivo* assays (sex-linked recessive lethal mutations in spermatocytes of *Drosophila melanogaster*) (Fahrig, 1974; Fahrig *et al.* cited in ATSDR, 2001; Vogel and Chandler cited in WHO, 1987) and *in vitro* studies on gene mutations in *Salmonella typhimurium* (NTP, 1999; Donnelly *et al.*, Markiewicz *et al.*, Simmon *et al.*, Waters *et al.* cited in ATSDR, 2001) and *Escherichia coli* (Andersen *et al.* cited in WHO, 1987; Lemma and Ames, Moriya *et al.*, Simmon *et al.*, Waters *et al.* cited in ATSDR, 2001).

Studies on DNA damage were also negative for *Escherichia coli* (Fahrig cited in ATSDR, 2001), *Bacillus subtilis* (Waters *et al.* cited in ATSDR, 2001), Chinese hamster ovary cells (Ehrlich cited in ATSDR, 2001), Chinese hamster V79 cells (Dahlhaus *et al.* cited in ATSDR, 2001), and mouse embryonic fibroblast cells (Wang and Lin cited in ATSDR, 2001).

A marginal increase in chromosomal aberrations (dicentrics and acentrics) was reported by Bauchinger *et al.* (cited in HSDB, 2002). Evidence of chromosomal aberrations in *in vitro* studies of human lymphocytes and Chinese hamster ovary cells and sister chromatid exchange in Chinese hamster ovary cells was reported by NTP (1999). Assays in *Saccharomyces cerevisiae* reported positive results (Fahrig, Fahrig *et al.*, Waters *et al.* cited in ATSDR, 2001) and negative results (Fahrig *et al.* cited in ATSDR, 2001) for recombination, and positive results for induction of gene mutations (Fahrig *et al.* cited in ATSDR, 2001).

PCP is a weak inducer of DNA damage in the absence of metabolic activation; however, with metabolic activation prophase induction and DNA strand breaks do occur in human lymphocytes (likely via oxygen radicals). Most bacterial assays and *in vitro* assays with mammalian cells did not indicate the potential for gene (point) mutations, although chromosomal aberrations were induced by PCP exposure in mammalian cells (*in vitro*) and in lymphocytes of exposed persons (*in vivo*). Genotoxicity of PCP may be mediated through the metabolite tetrachlorohydroquinone (capable of binding to DNA and producing strand breaks); however, this metabolite has not been detected in exposed humans *in vivo* (Seiler cited in HSDB, 2002).

4.3 Acute Effects

Pentachlorophenol has been widely used as an herbicide, insecticide, fungicide, and wood preservative. The acute effects observed in humans are very similar to those reported in experimental animal studies; however, there is no information on actual human exposure, limiting the ability to establish dose-response relationships (WHO, 1987).

4.3.1 Acute Human Effects

Cases of acute human poisoning typically involved inhalation or dermal contact with pentachlorophenol in wood preservatives or herbicides. Individuals were exposed within the agriculture and wood treatment industries and through home and garden use (home and garden use is now restricted). These cases involved exposure to technical grade pentachlorophenol, which contains known impurities of polychlorinated dibenzo-*p*-dioxins (PCDD) and

dibenzofurans (PCDF) (WHO, 1987; ATSDR, 2001). None of the acute human exposure studies identified adequately described pentachlorophenol exposure concentrations or duration.

Acute effects reported in humans exposed via inhalation to technical grade pentachlorophenol include, irritation of exposed epithelial tissue (eyes, nose, throat), extreme weakness, a rise in metabolic rate, elevated body temperature (hyperthermia) and effects on the central nervous system, cardiovascular system, liver and blood (WHO, 1987; ATSDR, 2001). Chloracne associated with exposure to technical grade pentachlorophenol has been associated with the micro-contaminant PCDD (WHO, 1987; ATSDR, 2001).

For example, the lethal effects of acute occupational exposure (inhalation and dermal) to PCP in wood preservatives, herbicides and fungicides was attributed to hyperthermia caused by the uncoupling of mitochondrial oxidative phosphorylation (Bergner *et al.*, Gordon cited in WHO, 1987; Menon cited in ATSDR, 2001; Gray *et al.*, 1985). The clinical findings in the case of one fatality (after three weeks exposure to PCP dust in a chemical plant) were consistent with hyperthermia. Cardiovascular and hematological effects were reported, cerebral edema and fatty degeneration of the liver and lungs were noted on necropsy (Gray *et al.*, 1985). The exposure concentrations for these situations were unknown.

Non-lethal effects reported in workers acutely exposed to pentachlorophenol dust at concentrations greater than 1 mg/m^3 (0.09 ppm) included severe irritation of the eyes and upper respiratory tract (accompanied by violent sneezing and coughing). Irritation of mucous membranes (nose, throat, eyes) occurred at 0.3 mg/m^3 (0.03 ppm) (Deichmann and Keplinger, 1981; ACGIH, 1986; NIOSH, 1996). Sensitivity was decreased in workers conditioned to exposure, who could tolerate concentrations up to 2.4 mg/m^3 (0.2 ppm). Based on this information, the National Institute of Occupational Safety and Health (NIOSH) have determined an immediately dangerous to life and health (IDLH) exposure limit of 2.5 mg/m^3 (0.2 ppm) for pentachlorophenol (NIOSH, 1996). It should be noted that these effects were reported for healthy workers and likely overestimate the exposure at which effects might occur in the general public. There are a number of limitations to be considered when using data from people exposed in the work place. Generally, the person exposed is a healthy, young to middle aged, male adult; concurrent exposures to other chemicals are very likely; and, the exposure concentrations are often difficult to define. The occupational IDLH value and acute effects concentrations for occupational exposures are presented in Table 4.

There is one case of acute poisoning (suicide) by ingestion of pentachlorophenol, however, the ingested dose was unknown (Cretny cited in ATSDR, 2001). A lethal dose of approximately 17 mg/kg was estimated for human ingestion of pentachlorophenol (purity not specified) by Driesbach (cited in ATSDR, 2001).

Table 4 Effects Associated with Acute Inhalation Exposure (Occupational) to Pentachlorophenol (Humans)

Effects Reported	Exposure Period	Air Concentration mg/m ³ (ppm) ^a	Reference
Immediately dangerous to life and health	8 hour time-weighted average (occupational)	2.5 (0.2)	NIOSH, 1996
Severe effects on eyes and upper respiratory tract	8 hour time-weighted average (occupational)	1 (0.09)	Deichmann and Keplinger, 1981; ACGIH, 1986; NIOSH, 1996
Severe irritation to nose, throat and eyes	8 hour time-weighted average (occupational)	0.3 (0.03)	Deichmann and Keplinger, 1981; ACGIH, 1986; NIOSH, 1996

^a 1 ppm = 10.9 mg/m³. Based on PC MW of 266.32 and the formula: mg/m³ = ppm x (MW/24.45) for chemicals in air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

4.3.1.1 Children Susceptibility and Acute Exposure Effects

Many factors can increase children's susceptibility to the toxic effects of chemicals; their vulnerability is often related to physiology and developmental stage. Enzyme systems are involved in pentachlorophenol toxicity (mitochondrial oxidative phosphorylation) and metabolism by the liver. The activity levels of these enzymes vary at different stages of development and may be responsible for an observed greater sensitivity of young children to pentachlorophenol exposure (ATSDR, 2001).

Studies on infants and children tend to demonstrate similar acute exposure effects experienced by adults (hyperthermia associated with the uncoupling of oxidative phosphorylation), and suggest a greater susceptibility of children to pentachlorophenol toxicity (Robson *et al.*, Smith *et al.*, Chapman and Robson cited in ATSDR, 2001; Hayes cited in Fisher, 1991).

Nine newborn infants exposed to pentachlorophenol in diapers and nursery linens (antimildew agent) showed severe symptoms of hyperthermia (high fever, profuse sweating, increased respiration, labored breathing, tachycardia, and hepatomegaly), irritability, lethargy, metabolic acidosis, proteinuria, increased blood urea nitrogen, and pneumonia or bronchiolitis. Pentachlorophenol was detected in blood and urine samples. Two of the infants died, autopsy revealed hepatic (fatty metamorphosis) and renal (fatty vacuolar changes in the renal tubules) effects (Robson *et al.*, Smith *et al.* cited in ATSDR, 2001). The exposure concentrations for these situations were unknown.

A three-year-old child exposed to pentachlorophenol contaminated bath water also experienced symptoms associated with hyperthermia, however, no signs of hyperthermia were reported in older children or adult family members bathing in the same contaminated water (Chapman and Robson cited in ATSDR, 2001). The exposure concentration for this situation was unknown. In cases of acute, fatal poisonings, the levels of pentachlorophenol detected in various organs and fluids of children and infants were lower than those detected in adults (Hayes cited in Fisher, 1991).

4.3.1.2 Respiratory Effects

Inhalation of PCP at air concentrations greater than 0.3 mg/m^3 (0.03 ppm) is associated with violent sneezing and coughing as well as severe irritation of the upper respiratory tract (Deichmann and Keplinger, 1981; ACGIH, 1986; NIOSH, 1996). Other respiratory effects and symptoms associated with acute inhalation of PCP include fatty degeneration of the lungs (Gray *et al.*, 1985), accelerated respiration and labored breathing (Robson *et al.*, Smith *et al.* cited in ATSDR, 2001).

4.3.2 Acute and Sub-Acute Animal Effects

Animal data for the acute effects of inhalation exposure to pentachlorophenol were limited to two studies of lethal air concentrations (LC_{50} values) for sodium pentachlorophenol in rats (Hoben *et al.* cited in WHO, 1987) and mice (Demidenko cited in WHO, 1987). The available LC_{50} values are presented in Table 5.

Table 5 Lethal Concentrations for Acute Inhalation Exposure to Pentachlorophenol (Experimental Animals)

Effects Reported	Exposure Period	Air Concentration mg/m^3 (ppm) ^a	Species	Reference
Death (LC_{50})	45 minutes	14 (1.3)	Rats	Hoben <i>et al.</i> cited in ATSDR, 2001.
Death (LC_{50})	Not reported	355 (32.6)	Rat	Demidenko cited in WHO, 1987.
Death (LC_{50})	Not reported	225 (20.6)	Mouse	Demidenko cited in WHO, 1987.

^a 1 ppm = 10.9 mg/m^3 . Based on PC MW of 266.32 and the formula: $\text{mg/m}^3 = \text{ppm} \times (\text{MW}/24.45)$ for chemicals in air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

Acute oral exposure studies reported LC_{50} values ranging from 80 to 120 mg/kg in rats and 117 to 177 mg/kg in mice (St. Omer and Gadusek cited in ATSDR, 2001; Borzelleca *et al.* cited in WHO, 1987; Renner *et al.* cited in ATSDR, 2001). Age-related sensitivity was reported for rats, with the lowest of the LD_{50} ranges being 50 mg/kg for preweaned rats, 80 mg/kg for adult rats and 220 mg/kg for juvenile rats (St. Omer and Gadusek cited in ATSDR, 2001). Based on these results and the LC_{50} identified for rats (14 mg/m^3 or 11.7 mg/kg) by Hoben *et al.*, (cited in WHO, 1987), sodium pentachlorophenol is significantly more toxic via inhalation than ingestion (WHO, 1987).

Two studies on sub-acute effects in animals following inhalation were identified (Ning *et al.* cited in WHO, 1987; Demidenko cited in WHO, 1987). The NOAELs and LOAEL's associated with the effects reported in these studies are presented in Table 6.

Table 6 NOAELs and LOAELs for Sub-acute Inhalation Exposure to Pentachlorophenol (Experimental Animals)

Effects Reported	Exposure Period	Air Concentration mg/m ³ (ppm) ^a	Species	Reference
Haematological:				
LOAEL	4hr/d,	21.4 (1.9)	Rabbits	Ning <i>et al.</i> cited in WHO, 1987.
NOAEL	6d/wk,	3.1 (0.3)		
Increased serum-gamma-globulin, hyperglycaemia	4 mo			
NOAEL	4hr/d,	21.4 (1.9)	Rabbits	Ning <i>et al.</i> cited in WHO, 1987.
Increased alpha-globulin, beta-globulin or serum albumin	6d/wk,			
	4 mo			
LOAEL	4hr/d,	2.97 (0.3)	Rats and Rabbits	Demidenko cited in WHO, 1987.
Anemia, leukocytosis, eosinophilia, hyperglycaemia	4 mo			
Lung:				
LOAEL	4hr/d,	21.4 (1.9)	Rats and Rabbits	Ning <i>et al.</i> cited in WHO, 1987.
NOAEL	6d/wk,	3.1 (0.3)		
Increased lung weight	4 mo			
Liver:				
LOAEL	4hr/d,	3.1 (0.3)	Rabbits	Ning <i>et al.</i> cited in WHO, 1987.
Increased liver weight	6d/wk,			
	4 mo			
LOAEL	4hr/d,	21.4 (1.9)	Rats	Ning <i>et al.</i> cited in WHO, 1987.
NOAEL	6d/wk,	3.1 (0.3)		
Increased liver weight	4 mo			
LOAEL	4hr/d,	2.97 (0.3)	Rats and Rabbits	Demidenko cited in WHO, 1987. ^b
Dystrophic processes in liver	4 mo			
Kidney:				
LOAEL	4hr/d,	21.4 (1.9)	Rats	Ning <i>et al.</i> cited in WHO, 1987.
NOAEL	6d/wk,	3.1 (0.3)		
Increased kidney weight	4 mo			
Adrenal gland:				
LOAEL	4hr/d,	21.4 (1.9)	Rats	Ning <i>et al.</i> cited in WHO, 1987.
NOAEL	6d/wk,	3.1 (0.3)		
Increased adrenal gland weight	4 mo			

^a 1 ppm = 10.9 mg/m³. Based on PC MW of 266.32 and the formula: mg/m³ = ppm x (MW/24.45) for chemicals in air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

The World Health Organization (1987) has indicated that pentachlorophenol is more toxic via the inhalation versus oral route of exposure. Kunde and Böhme (cited in WHO, 1987) used the LOAEL concentration of 2.97 mg/m³ (liver and blood effects in rats and rabbits) reported by Demidenko (cited in WHO, 1987) to calculate an effective daily dose of 0.3 mg/kg body weight per day for rats (assuming 100% uptake and absorption). In a recent summary of sub-acute oral studies in rats (from 28 days to 8 months), the lowest comparable LOAEL value (for increased liver and kidney weights) was 2 mg/kg body weight per day for >99% pure pentachlorophenol given twice a week for 28 weeks (Blakley *et al.* cited in ATSDR, 2001), still nearly 10 times higher than the calculated dose received via inhalation.

4.3.2.1 Respiratory Effects

An increased lung weight was reported in rats and rabbits following sub-acute (four months) inhalation exposure to PCP (Ning *et al.* cited in WHO, 1987)

4.3.2.2 Haematological Effects

Hematological effects reported in rabbits following four months PCP inhalation exposure included increased serum-gamma-globulin, hyperglycemia, increased alpha-globulin, beta-globulin or serum albumin (Ning *et al.* cited in WHO, 1987). Demidenko (cited in WHO, 1987) reported anemia, leukocytosis, eosinophilia and hyperglycemia in rats and rabbits exposed via inhalation over the same time period.

4.3.2.3 Hepatic and Renal Effects

An increase in liver weights of rats and rabbits, an increase in kidney weight of rats (Ning *et al.* cited in WHO, 1987) and dystrophic processes in the liver of rats and rabbits (Demidenko cited in WHO, 1987) were reported following four months inhalation exposure.

4.3.2.4 Other Effects

An increased adrenal gland weight was reported for rats exposed to PCP via inhalation over a four month period (Ning *et al.* cited in WHO, 1987).

4.4 Chronic Effects

4.4.1 Chronic Human Effects

Much of the quantitative information regarding inhalation of PCP is from occupational studies, where the variability in employees makes it difficult to separate out short-term from long-term exposure duration. The majority of human inhalation exposure data available has been collected after occupational exposures. As indicated in Section 4.3.1, there are limitations to be considered when using data from people exposed in the work place.

Chronic non-occupational exposure to PCP was often associated with interior wood treated wood preservatives containing PCP or its sodium salt (WHO, 1987; ATSDR, 2001).

4.4.1.1 Children Susceptibility and Chronic Exposure Effects

Chronic developmental studies in animals (Section 1.3.2.2) indicate that pentachlorophenol affects thyroid function (decreases serum thyroxine levels) (Jekat *et al.*, 1994, Beard and Rawlings, Beard *et al.* cited in ATSDR, 2001). Decrements in intellectual function of children have been associated with thyroxine deficiency during prenatal and postnatal life (Bargagna *et al.*, Birrell *et al.*, Kooistra *et al.*, cited in ATSDR, 2001).

Where provided, PCP air concentrations (or ranges of concentrations) and effects reported following occupational exposures are summarized in Table 7. Occupational studies carry a high potential for confounding exposure factors that limit their interpretation; the actual exposures to PCP may not be represented by the ambient air concentrations provided.

Table 7 Effects Associated with Chronic Inhalation Exposure (Occupational) to Pentachlorophenol (Humans)

Effects Reported	Exposure Period	Air Concentration $\mu\text{g}/\text{m}^3$ (ppb) ^a	Reference
Liver Effects:			
Elevated activities of serum-aminotransferases and alpha-glutamyl transpeptidase	3 years (avg) 0.5 to 12 years (range)	2.4 (0.22) (avg) 0.3 to 8 (0.028 - 0.73) (range)	Zober <i>et al.</i> cited in WHO, 1987.
Hepatotoxic effects in humans	Chronic	0.8 (0.073)	Hassauer <i>et al.</i> cited in RIVM 2001.
Neurological Effects:			
No effects on motor or sensory nerve conduction velocities	16 years (avg) 4 to 24 years (range)	0.3 to 180 (0.028 to 17) (range)	Triebig <i>et al.</i> , 1987
Genotoxic Effects:			
No effect on chromosomal aberrations or sister chromatid exchange	3 to 34 years (range)	1.2 to 180 (0.11 to 17) (range)	Ziensen <i>et al.</i> cited in HSDB, 2002.

^a 1 ppb = 10.9 $\mu\text{g}/\text{m}^3$. Based on PC MW of 266.32 and the formula: $\text{mg}/\text{m}^3 = \text{ppm} \times (\text{MW}/24.45)$ for chemicals in air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

4.4.1.2 Respiratory Effects

Chronic inhalation exposure to PCP in the workplace has been associated with inflammation of the upper respiratory tract and bronchitis (Baader and Bauer, Klemmer *et al.* cited in WHO, 1987). Confounding factors in these studies include exposure to particulate matter and other compounds in workplace air that could cause the same effects (i.e., dieldrin, chromium, fluorine, arsenic, copper, boron, and tin compounds).

4.4.1.3 Neurological Effects

A significant association was reported between PCP blood levels and objective neurological test results (reading speed, naming speed, paired associated learning, visual short-term memory) for 15 women following exposure to wood preserving chemicals (no air concentrations provided). These results are limited by the small number of subjects examined and concurrent exposure to high levels of lindane and other solvents (Peper *et al.* cited in ATSDR, 2001).

A reduction in nerve conduction velocity was associated with chronic exposure (12 year mean) to pentachlorophenol in a chemical processing factory. Pentachlorophenol levels were measured in plasma (0.02-1.5 $\mu\text{g/L}$) and urine (13-1224 $\mu\text{g/L}$) (Triebig *et al.* cited in HSDB, 2002). No effects on motor or sensory nerve conduction velocities were detected in a longitudinal study of 10 workers chronically exposed (4 to 24 years, average of 16 years) to technical grade pentachlorophenol at air concentrations ranging from 0.3 to 180 $\mu\text{g/m}^3$. Pentachlorophenol levels were measured in serum (38-1270 $\mu\text{g/L}$) and urine (8-1224 $\mu\text{g/L}$) (Triebig *et al.*, 1987).

A health survey of occupational exposure during pentachlorophenol production detected a significant association with a reduction in motor nerve conduction velocity with a subgroup working in the trichlorobenzene tank area and concurrently exposed to pentachlorophenol and chlorinated dibenzo- p-dioxins (CDDs) (Cheng *et al.* cited in ATSDR, 2001).

In a case-control study, Parkinson's disease was significantly associated with >15 years exposure to wood paneling in the home, contact with wood preservatives in free time, and contact with wood preservatives at work (Seidler *et al.* cited in ATSDR, 2001). These results were limited by an increased potential of the Parkinson's patients to have confounding exposures to organochlorines, alkylated phosphates/carbamates, heavy metals, solvents, exhaust fumes and carbon monoxide.

4.4.1.4 Haematological Effects

Klemmer *et al.* (cited in WHO, 1987) reported an increase in immature leukocytes and basophiles in workers chronically exposed to technical-grade pentachlorophenol. Elevated urinary excretion of coproporphyrins (Hryhorczuk *et al.* cited in ATSDR, 2001), porphyrin and delta-amino levulinic (Cheng *et al.* cited in ATSDR, 2001) were reported in epidemiological studies of exposed pentachlorophenol production workers. Exposure concentrations for these situations were unknown.

4.4.1.5 Liver Effects

Altered liver function (elevated activities of serum-aminotransferases and alpha-glutamyl transpeptidase) was reported in an evaluation of a 23 woodworkers involved in PCP application and exposed to an average PCP air concentration of 2.4 $\mu\text{g/m}^3$ (range from 0.3 to 8 $\mu\text{g/m}^3$). Pentachlorophenol levels were measured in urine (46 $\mu\text{g PCP/g creatine}$) and plasma (1 $\mu\text{g/mL}$) (Zober *et al.* cited in WHO, 1987).

An inhalation criteria of 0.04 $\mu\text{g/m}^3$ was recommended to the UBA (Germany) for chronic inhalation exposure to pentachlorophenol, based on a LOAEC of 0.8 $\mu\text{g/m}^3$ for hepatotoxic

effects in humans (source document not identified) and an uncertainty factor of 20 (Hassauer *et al.* cited in RIVM, 2001).

4.4.1.6 Immunological Effects

Immunological effects reported in 38 individuals exposed (1 to 13 years) to pentachlorophenol in treated log homes included: activated T-cells, autoimmunity, immunosuppression, and B-cell dysregulation (McConnachie and Zahalsky cited in ATSDR, 2001). No air concentrations were reported, but the mean serum levels of PCP in individuals still living in log homes were higher (884 $\mu\text{g/L}$) than those previously reported (Cline *et al.* cited in ATSDR, 2001) for individuals living in log homes (420 $\mu\text{g/L}$) and the general public (40 $\mu\text{g/L}$).

Daniel *et al.*, (1995) concluded that severe T lymphocyte dysfunction was associated with increased blood levels of pentachlorophenol, following the examination of immune parameters (lymphocyte populations, *in vitro* responses to mitogenic and allogenic stimulation, plasma neopterin levels, plasma cytokine and cytokine receptors) and pentachlorophenol levels in the blood of 188 patients exposed to pentachlorophenol-containing pesticides for greater than six months (exposure concentrations unknown).

4.4.1.7 Endocrine Effects

Chemicals possessing the ability of chemicals to mimic or block endogenous hormones, or otherwise interfere with the normal function of the endocrine system are now commonly referred to as endocrine disruptors. By the nature of their impact on natural hormones in the body, endocrine disruptors have adverse reproductive, immunological and neurobehavioral effects (ATSDR, 2001).

Using an *in vitro* test system, Tran *et al.* (cited in ATSDR, 2001) demonstrated the ability of pentachlorophenol (99% pure) to inhibit human progesterone activity by competitively binding to the human progesterone receptor.

Elevated blood levels of pentachlorophenol in a group of women with reproductive disorders were associated with adrenocortical insufficiency and thyroid dysfunction (Gerhard *et al.* cited in ATSDR, 2001). In a study of a group women with repeated miscarriages Gerhard *et al.* (cited in ATSDR, 2001) found an inverse correlation between pentachlorophenol and triiodothyronine serum levels. This correlation was also reported for another cohort of women with gynecological and/or endocrinological disorders (Gerhard *et al.*, 1999), as well as an increased incidence of euthyroid goitre and alterations in endocrine hormones. These studies could not draw a causal relationship with pentachlorophenol exposure due to confounding exposure to other chlorinated hydrocarbons (ATSDR, 2001).

4.4.1.8 Developmental Effects

One study correlated the incidence of congenital eye cataracts in the off-spring of male sawmill workers exposed to a mixture of the sodium salts of pentachlorophenol and tetrachlorophenol (Dimich-Ward *et al.* cited in ATSDR, 2001).

4.4.1.9 Reproductive Effects

Elevated blood levels of pentachlorophenol were reported in a group of women with histories of spontaneous abortions, infertility and menstrual disorders (Gerhard *et al.* cited in ATSDR, 2001) and in women with repeated miscarriages (Gerhard *et al.* cited in ATSDR, 2001). The women were exposed to wood preservatives (PCP and other chlorinated hydrocarbons) in their homes via off gassing and dermal contact. Pentachlorophenol was highest in the blood of women with infertility disorders. These studies involved confounding exposure to other chlorinated hydrocarbons (ATSDR, 2001).

In studies on males with decreased sperm density, PCP was observed to be selectively concentrated in human seminal plasma at concentrations ranging from 100 to 200 ppb (Dougherty *et al.*, Kuehl and Dougherty cited in WHO, 1987).

4.4.1.10 Carcinogenic Effects

Several epidemiological studies, including studies from Finland and New Zealand, did not detect any association of occupational inhalation exposure of pentachlorophenol with cancer incidence (IARC, 1991; Jäppinen *et al.*, Johnson *et al.*, 1990, Robinson *et al.* cited in ATSDR, 2001). However, epidemiological studies from Canada, Sweden, New Zealand and the United States have associated occupational exposure to mixtures of chlorophenols (including PCP) with an increased incidence of soft tissue sarcomas, nasal and nasopharyngeal cancers and non-Hodgkin lymphoma (IARC, 1999).

Associations between soft tissue sarcoma and exposure to technical-grade pentachlorophenol have been determined by several authors in their analysis of case-control and group studies (Eriksson *et al.*, Hardell *et al.*, Hoppin *et al.* cited in ATSDR, 2001). Two studies reported associations between the incidence of non-Hodgkin's lymphoma and pentachlorophenol exposure (Hardell *et al.*, Hertzman *et al.* cited in ATSDR, 2001). One study associated mortality from kidney cancer with exposure to technical grade pentachlorophenol (Ramlow *et al.* cited in ATSDR, 2001). All of the epidemiological studies on the carcinogenic effects of occupational exposure to pentachlorophenol were lacking in specific exposure characterization; involved exposures to other confounding chemicals; and, had small case numbers with limited statistical power (WHO, 1987; ATSDR, 2001).

The International Agency for Research on Cancer (IARC) and the US Environmental Protection Agency have classified pentachlorophenol as a probable human carcinogen based on limited evidence in humans and sufficient evidence in animals (IARC, 1991; IRIS, 1993).

4.4.1.11 Other Effects

Numerous reports of skin abnormalities (rashes, cysts, and chloracne) were associated with occupational and residential exposure to pentachlorophenol and sodium pentachlorophenate (Baader and Bauer, Klemmer *et al.* cited in WHO, 1987; Hosenfeld *et al.*, Seghal and Ghorpade, Cheng *et al.*, Hryhorczuk *et al.*, O'Malley *et al.* cited in ATSDR, 2001). However, these dermal effects were more likely due to exposure to contaminants known to induce chloracne (chlorinated dibenzo- p-dioxins, dibenzofurans) rather than pentachlorophenol (ATSDR, 2001).

4.4.2 Chronic Animal Effects

In the data reviewed, no chronic animal PCP inhalation studies were identified. Due to the lack of inhalation data, several key ingestion studies were identified. Examples of the lowest NOAELs and LOAELs for liver and kidney, endocrine, reproductive, developmental and carcinogenic effects are briefly summarized below in Table 8. The use of pure or technical grade pentachlorophenol was documented where the information was provided.

Table 8 Examples of NOAELs and LOAELs Associated with Chronic Ingestion of Pentachlorophenol (Experimental Animals)

Effects Reported	Exposure Period	Dose Rate (mg/kg body weight/day)	Species	Reference
Liver and Kidney Effects:				
NOAEL	2 years	3 technical grade (90% pure)	rat	Schwetz <i>et al.</i> cited in ATSDR, 2001.
Increase in relative liver and kidney weights	28 day	2 (>99% pure)	rat	Blakley <i>et al.</i> cited in ATSDR, 2001.
Endocrine Effects:				
LOAEL Significantly decreased serum thyroxine concentrations	3 weeks prior to mating and throughout gestation and lactation	1	mink	Beard and Rawlings cited in ATSDR, 2001.
Developmental Effects:				
LOAEL Delayed ossification of the skull	9 days (gestation)	5 (97.5% pure)	rats	Schwetz <i>et al.</i> cited in ATSDR, 2001.
Reduced pup weight	70 days pre-mating through gestation and lactation	10	rats	Bernard <i>et al.</i> cited in ATSDR, 2001.
Impaired development of reproductive system	70 days pre-mating through gestation and lactation	60	rats and rabbits	Bernard <i>et al.</i> cited in ATSDR, 2001.

Table 8 Examples of NOAELs and LOAELs Associated with Chronic Ingestion of Pentachlorophenol (Experimental Animals) (continued)

Effects Reported	Exposure Period	Dose Rate (mg/kg body weight/day)	Species	Reference
Reproductive Effects:				
NOAEL No histological alterations in reproductive tissues	2 years	30 (99% pure)	rats	Chhabra <i>et al.</i> , 1999, NTP cited in ATSDR, 2001.
NOAEL No significant reproductive effects	5 weeks prior to mating and throughout gestation & lactation	1	sheep	Beard <i>et al.</i> cited in ATSDR, 2001.
LOAEL Decreases in the proportion of mated females accepting a second mating and mink that whelped; increased severity of cystic uterine glands	3 weeks prior to mating and throughout gestation & lactation	1	mink	Beard <i>et al.</i> cited in ATSDR, 2001.
Carcinogenic Effects:				
LOAEL Significant increases in the incidence of hemangiosarcomas, liver adenomas and carcinomas, and adrenal gland pheochromocytomas	2 years	17.5 technical grade (90% pure)	mice	NTP cited in ATSDR, 2001.
LOAEL Mesotheliomas and nasal squamous cell carcinomas	52 weeks	60 (99% pure)	rats	Chhabra <i>et al.</i> , NTP cited in ATSDR, 2001.

4.4.2.1 Liver and Kidney Effects

A chronic NOAEL of 3 mg/kg body weight/day (mg/kg/day) was identified in rats (no adverse effects on liver or kidneys) following two years exposure to technical grade (90% pure) pentachlorophenol (Schwetz *et al.* cited in ATSDR, 2001). This study was the basis for the US EPA chronic oral RfD of 3 µg/kg/day (IRIS, 1993).

Blakley *et al.* (cited in ATSDR, 2001) reported an increase in relative liver and kidney weights in rats exposed to 2 mg/kg/day pure (>99%) pentachlorophenol, twice a week over a 28 day period. This effective dose for pure pentachlorophenol was lower than the NOAEL previously identified by Schwetz *et al.* (cited in ATSDR, 2001) for technical grade pentachlorophenol.

4.4.2.2 Endocrine Effects

Pentachlorophenol has been shown to compete with the thyroxine binding site on transthyretin, a major thyroxine transport protein (den Besten *et al.* cited in ATSDR, 2001). Pentachlorophenol effects on thyroid function have been well documented in animal studies (ATSDR, 2001). The effects reported include decreased serum thyroxine concentration in orally exposed rats, sheep and mink (Beard and Rawlings, Beard *et al.*, Jekat *et al.*, van Raaij *et al.* cited in ATSDR, 2001), decreased thyroxine and triiodothyronine response to thyroid stimulating hormone in orally exposed sheep (Beard and Rawling cited in ATSDR, 2001), and decreased uptake of thyroxine into cerebrospinal fluid in orally exposed rats (van Raaij *et al.* cited in ATSDR, 2001).

Female guinea pigs exhibit cyclic and luteal similarities to humans. Estrogenic effects (statistically significant elevation of serum progesterone) were demonstrated on the reproductive tract of adult female guinea pig following 14 days (during most of the 16 day estrus cycle) subcutaneous exposure to 40 mg/kg/day purified pentachlorophenol. Pentachlorophenol was also demonstrated to inhibit (40%) estrogen receptor binding by radio labeled estradiol (Danzo *et al.*, 2001).

The ATSDR (2001) established a chronic-duration Minimum Risk Level (MRL) for pentachlorophenol based on the potential for alterations in thyroid hormone levels (significantly decreased serum thyroxine concentrations) and decreased relative thyroid weight. A chronic MRL of 1 µg/kg/day was established based on a LOAEL of 1 mg/kg/day for thyroid effects reported in mink exposed in a multigeneration study for three weeks prior to mating and throughout gestation and lactation (Beard and Rawlings cited in ATSDR, 2001).

The National Institute of Public Health and the Environment (RIVM), under the Dutch soil protection act, established a tolerable daily intake (TDI) of 3 µg/kg/day based on the same chronic LOAEL of 1 mg/kg/day for thyroid effects in mink (RIVM, 2001).

4.4.2.3 Developmental Effects

Developmental effects in several rat studies with pure and technical grade pentachlorophenol may be associated with a disruption in endocrine function (ATSDR, 2001). Delayed ossification of the skull was reported in offspring of rats administered 5 mg/kg/day *pure* pentachlorophenol on gestational days 6-15 (Schwetz *et al.* cited in ATSDR, 2001). The same study reported developmental effects (resorptions, soft tissue and skeletal malformations, decreases in fetal body weight) in off-spring of rats ingesting 80 mg/kg/day *technical grade* PCP on gestational days 6-15. A NOAEL of 30 mg/kg/day was reported for technical grade PCP, indicating greater developmental toxicity of pure pentachlorophenol (Schwetz *et al.* cited in ATSDR, 2001).

A decrease in pup weight was reported in rats following maternal exposure to 10 mg/kg/day for 70 days pre-mating and throughout gestation and lactation (Bernard *et al.* cited in ATSDR, 2001). No developmental effects were observed in the first generation of rabbits exposed during gestation (days 6 to 18) to 30 mg/kg/day pentachlorophenol (89% pure) in a two-generation study (Bernard *et al.* cited in ATSDR, 2001). Impaired development of the reproductive system occurred in offspring of rats and rabbits for exposed for 70 days pre-mating and throughout gestation and lactation at a dose of 60 mg/kg/day. (Bernard *et al.*, Bernard *et al.* cited in ATSDR, 2001).

4.4.2.4 Reproductive Effects

Male and female rats orally exposed to 30 mg/kg bd wt/day pure pentachlorophenol for two years demonstrated no histological alterations in reproductive tissues (Chhabra *et al.* cited in ATSDR, 2001; NTP 1999).

No significant reproductive effects were observed in sheep exposed to 1 mg/kg bd wt/day pentachlorophenol for five weeks prior to mating and throughout gestation and lactation (Beard *et al.* cited in ATSDR, 2001).

Reproductive effects in mink (decreases in the proportion of mated females accepting a second mating and mink that whelped) occurred following exposure to 1 mg/kg bd wt/day pentachlorophenol for three weeks prior to mating and throughout gestation and lactation. Maternal exposure also increased the severity of cystic uterine glands (Beard *et al.* cited in ATSDR, 2001). The ATSDR (2001) recommended an intermediate-duration MRL of 1 μ g/kg/day for pentachlorophenol based on the results of this study.

4.4.2.5 Carcinogenic Effects

The International Agency for Research on Cancer (IARC) and the US Environmental Protection Agency have classified pentachlorophenol as a probable human carcinogen based on sufficient evidence of carcinogenicity in orally exposed animals (IARC, 1991; IRIS, 1993).

Chronic ingestion studies of pentachlorophenol exposure were carried out in mice and rats by the National Toxicology Program (NTP, 1989; 1999). The carcinogenicity of technical grade pentachlorophenol (90% pure) was clearly demonstrated in mice with significant increases in the incidence of hemangiosarcomas, liver adenomas and carcinomas, and adrenal gland pheochromocytomas (LOAEL of 17.5 mg/kg/day) (NTP, 1989; ATSDR, 2001). This data was identified by IRIS in the development of their oral cancer slope factor (risk estimate) for pentachlorophenol. (IRIS, 1993).

Carcinogenic effects in rats following 52 weeks exposure to pure pentachlorophenol (99%) included mesotheliomas and nasal squamous cell carcinomas (LOAEL of 60 mg/kg/day) (Chhabra *et al.* cited in ATSDR, 2001; NTP, 1999; ATSDR, 2001).

4.5 Summary of Adverse Health Effects of Pentachlorophenol

Pentachlorophenol (PCP) is highly toxic, regardless of the route, length, and frequency of exposure. It is readily absorbed through cell membranes of the lungs, gastrointestinal tract and skin. Once absorbed, PCP is distributed to the adipose tissue, liver, lungs, kidneys, blood, brain and spleen. There is evidence for long-term accumulation and storage of pentachlorophenol, with higher concentrations detected in the liver and kidney, and lower concentrations in body fat, brain and muscle tissue. Chromosomal aberrations have been reported in human lymphocytes and Chinese hamster ovary cells.

The acute effects of PCP reported in humans include, irritation of exposed epithelial tissue (eyes, nose, throat), a rise in metabolic rate, hyperthermia and effects on the central nervous system,

respiratory system, cardiovascular system, liver and blood. Studies on infants and children suggest a greater susceptibility of children to acute pentachlorophenol toxicity. Acute and sub-acute inhalation exposure of animals to pentachlorophenol has been reported to affect the respiratory system, blood, liver, kidney and adrenal system. Comparisons of effects in acute inhalation and oral studies on rats and rabbits indicate that pentachlorophenol is significantly more toxic via inhalation than ingestion.

In humans, chronic PCP inhalation has been associated with effects on the respiratory system, endocrine system, central nervous system and reproductive system. Adverse effects on the liver and blood were also reported. No chronic inhalation animal studies were identified. Chronic animal ingestion studies reported: effects on the liver and kidney; endocrine and reproductive system effects; developmental effects; and, carcinogenicity. Results of these studies suggest that, for the same species and endpoint evaluated, the purified form of PCP is more toxic than the technical formulation (i.e., liver and kidney effects in rats).

The lowest adverse effects levels were reported for reproductive effects (mink) followed by liver and developmental effects. The International Agency for Research on Cancer and the US Environmental Protection Agency have classified pentachlorophenol as a probable human carcinogen based on limited evidence in humans and sufficient evidence in animals and cancer incidence associated with occupational exposures.

4.6 Vegetation

The primary impacts of air pollution on ecosystems result from the deposition onto or entry into plants, soil and water. Since plants are sessile organisms with large biologically active surface areas, they may be important receptors of airborne pollutants and exposure-related effects. This has been shown to be the case for primary air pollutants, e.g. sulphur dioxide (Cape, 1993). However, no published literature could be found on the direct effects through the atmosphere of pentachlorophenol on vegetation (plants).

Hulzebos *et al.* (1993) investigated the effects of pentachlorophenol on growth of lettuce in two soils and solution. It was found that soils with higher clay content had a higher EC₅₀ value for phenol compared to pentachlorophenol. The EC₅₀ represented the median effective concentration for decreased plant growth. Gunther and Pestemer (1990) reported levels of pentachlorophenol causing reduced fresh weight of shoots of oats (*Avena sativa*) after 14 days of exposure and turnips (*Brassica rapa*) after 10 days of exposure in a sandy loam soil. The EC₅₀s for oats and turnips were 20 and 10 mg/kg, respectively.

Surrogate information indicative of the relative amount of airborne pentachlorophenol that would have to deposit and accumulate in surface soil to pose adverse effects to terrestrial plants was also examined. Efroymson *et al.* (1997) developed toxicological benchmarks for screening environmental pollutants of potential concern for effects on terrestrial plants. These authors derived benchmark concentrations in soil as indicators of potential concern for effects on terrestrial plants. A relatively low benchmark concentration in soil suggests that the potential for the role of air pollution deposition to be a contributing factor to terrestrial plant toxicity may be more important. Whereas, a relatively high soil benchmark concentration in soil suggests that

the potential for the role of air pollution deposition to be a contributing factor to terrestrial plant toxicity is less important.

The screening benchmark concentration for phytotoxicity (plant damage) of pentachlorophenol was reported as 3 mg/kg soil (Efroymson *et al.*, 1997). The screening benchmark concentrations in soil for phytotoxicity of phenol and toluene were 70 and 200 mg/kg respectively. The relatively lower soil benchmark concentration for pentachlorophenol suggests that the potential for the role of air pollution deposition to be a contributing factor to terrestrial plant toxicity is more important than phenol and toluene via the soil pathway.

5.0 AIR SAMPLING AND ANALYTICAL METHODS

5.1 Reference Methods

Air sampling and analytical methods for pentachlorophenol used in practice by established agencies are reported. In general, standard air monitoring methods for pentachlorophenol are based on high and low volume solid sorbent or pump-and-tube sampling approaches. Widely employed and accepted reference air monitoring methods for pentachlorophenol have been developed, tested and reported by the United States Environmental Protection Agency (US EPA), National Institute of Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA). Refer to Table 9 for a description of individual method advantages and disadvantages.

5.1.1 *US EPA Compendium Method TO-4A*

US EPA has developed two methodologies suitable for sampling ambient air for trace-level concentrations of pentachlorophenol. US EPA Compendium Method TO-4A describes the determination of pesticides and polychlorinated biphenyls (including pentachlorophenol) in ambient air using high volume polyurethane foam (PUF) sampling followed by gas chromatographic/multi-detector detection (GC/MD) (US EPA, 1999). The advantages of this method include: low detection limits, effective for a broad range of pesticides/PCBs, PUF is reusable, low blanks, and excellent collection and retention efficiencies. Disadvantages of this method include: breakdown of PUF adsorbent may occur with polar extraction of solvents, contamination of glassware may limit detection limits, loss of some semi-volatile organics during storage, extraneous organics may interfere, and difficulty in identifying individual pesticides and PCBs using some detectors.

In this method, a high volume sampler is used to draw ambient air through a sorbent cartridge containing PUF. Common pesticides and PCBs (including pentachlorophenol) are captured on the cartridge while major inorganic atmospheric constituents pass through (although some airborne particles can be partially retained). The high volume sampler is operated for 24 hours at a rate of 200 to 288 L/min to collect a total volume of 288 to 403 m³. After sampling, the cartridge is returned to the laboratory for analysis. Pentachlorophenol is extracted from the sorbent cartridge with 10% diethyl ether in hexane and the concentration is determined using gas chromatography with an electron capture detector (GE-ECD). The detection limit of this procedure depends on the nature of the analyte and the length of the sampling period but can range from 0.2 pg/m³ to 200 ng/m³.

5.1.2 *US EPA Compendium Method TO-10A*

US EPA Compendium Method TO-10A describes the determination of pesticides and polychlorinated biphenyls (including pentachlorophenol) in ambient air using low volume polyurethane foam (PUF) sampling followed by gas chromatographic/multi-detector detection (GC/MD) (US EPA, 1999). The procedure is similar to US EPA Compendium Method TO-4A except that a low volume sampler is used instead of a high volume sampler. The advantages of

this method include: easy field use, proven methodology, easy to clean, effective for a broad range of compounds, portability, and good retention of compounds. Disadvantages of this method include: detectors are subject to responses from a variety of compounds other than target analytes, PCBs, dioxins and furans may interfere, certain organochlorine pesticides are complex mixtures and can make accurate quantitation difficult, and may not be sensitive enough for all target analytes in ambient air.

In this method, a low volume sampler is used to draw ambient air through a sorbent cartridge containing PUF or PUF in combination with another solid sorbent. Common pesticides and PCBs (including pentachlorophenol) are captured on the cartridge while major inorganic atmospheric constituents pass through (although some airborne particles can be partially retained). The low volume sampler is operated for 4 to 24 hours at a rate of 1000 to 5000 mL/min to collect a total volume of 240 to 7200 L. After sampling, the cartridge is returned to the laboratory for analysis. Pentachlorophenol is extracted from the sorbent cartridge with 5% diethyl ether in hexane and the concentration is determined using gas chromatography with an electron capture detector (GE-ECD). The detection limit of this procedure depends on the nature of the analyte and the length of the sampling period but can range from 1 to 100 ng/m³.

5.1.3 NIOSH Method 5512

In addition to the air monitoring methods for pentachlorophenol developed by the US EPA, both the NIOSH and the OSHA have also developed methods for pentachlorophenol that are suitable for occupational, personal and area monitoring. The methodology currently used by the NIOSH to determine pentachlorophenol in air (NIOSH Method 5512) consists of collecting pentachlorophenol on a filter, transferring to a liquid bubbler, desorbing with methanol and analyzing by high pressure liquid chromatography (HPLC) using an ultraviolet (UV) detector (NIOSH, 1994). Sampling is conducted by using a personal sampling pump to draw air through a 37-mm cellulose ester membrane filter (0.8- μ m pore size) supported by a stainless steel screen in a three-piece filter holder followed by a 25-mL bubbler with 15 mL of ethylene glycol. The suggested flow rate is between 0.5 and 1.0 L/min and the recommended volume collected is between 48 and 480 L. The working pentachlorophenol concentration range for this method is 0.13 to 11 mg/m³ for a 180 L sample.

5.1.4 OSHA Method 39

OSHA has developed a fully validated method for the determination of pentachlorophenol that is suitable for occupational, personal and area monitoring. The methodology currently used by the OSHA to determine pentachlorophenol in air (OSHA Method 39) was developed to be rapid, sensitive and precise (OSHA, 1982). This method consists of collecting pentachlorophenol by drawing a known volume of air through two specially prepared XAD-7 sampling tubes connected in series. Samples are desorbed with methanol and analyzed by high performance liquid chromatography (HPLC) using an ultraviolet (UV) detector. Unlike the previous NIOSH sampling method which employs ethylene glycol, this method does not require the use of a cumbersome bubbler for sampling. The advantages of this method are that there are no known interferences and HPLC analysis of pentachlorophenol is rapid, precise, and sensitive.

Disadvantages of this procedure include the fact that the method has not been field-tested and the sampling tubes are not commercially available.

Sampling is conducted by drawing a known volume of air through two specially prepared XAD-seven adsorbent tubes which are connected in series using a personal sampling pump calibrated to within 5% of the recommended flow rate. A small glass fiber disc is placed ahead of the resin bed of the front tube to trap any aerosols present in the air. The second tube serves as a backup section in the unlikely event of breakthrough. The suggested flow rate is 0.2 L/min and the recommended volume collected is 48 L after a sampling time of 4 hours. The reliable detection limit of the overall procedure is 0.33 µg per sample (0.007 mg/m³) for a 48 L air sample. This is the amount of pentachlorophenol spiked on the sampling device that allows recovery of an amount equivalent to the detection limit of the analytical procedure. The reliable quantitation limit is 0.33 µg per sample (0.007 mg/m³). This is the smallest amount of pentachlorophenol that can be quantified within the requirements of a recovery of at least 75% and a precision of ± 25% or better.

5.2 Alternative, Emerging Technologies

Reports, journal articles, conference proceedings and other sources known to contain information on ambient measurement methods were reviewed to determine the current status of alternative and emerging technologies for pentachlorophenol. The results of the review indicate a general lack of technologies for the ambient monitoring of pentachlorophenol beyond the reference methods described earlier. A recent US EPA-sponsored survey reinforces this by pointing out the need for methods development for chemicals such as pentachlorophenol (Mukund *et al.*, 1995). Despite this need, a few examples of alternative and emerging technologies have been developed and reported.

Alternative technologies for the collection and analysis of pentachlorophenol have been reported that use modifications of the reference methods previously mentioned. The most notable modifications involve alternative types of solid sorbent cartridges used in conjunction with the accepted sampling and analytical approaches developed by the US EPA. For instance, instead of the PUF cartridge used by the US EPA, others have suggested using a PUF/XAD-2 resin sandwich (Cessana *et al.*, 1997) or a PUF/Tenax-GC sandwich cartridge (McConnell and Bidleman, 1998). There are no apparent advantages or disadvantages in using these alternative technologies over the conventional US EPA methods.

Some emerging technologies for the collection and analysis of pentachlorophenol have been described. These technologies are, however, more appropriate for special indoor studies and less for ambient monitoring. Schnelle-Kreis *et al.* (2000) and Meibner and Schweinsberg (1996) have described methods for collecting and analyzing passively deposited suspended particulate for the investigation of pentachlorophenol exposure from indoor air. In both cases, particulate was passively collected and pentachlorophenol was subsequently determined by gas chromatography with an electron capture detector (GC-ECD). These techniques are only useful in determining the particle-bound portion of the total indoor exposure to pentachlorophenol.

Wuske *et al.* (1998) describe a passive pentachlorophenol surface emission patch (PSEP) that has been developed to address the broad need for semi-quantitative, non-destructive measurement of pentachlorophenol emission rates from various surfaces. The patch consists of glass fiber material, which has been shown to be an excellent absorbent of gaseous pentachlorophenol. After several hours of passive exposure on an indoor surface the patch is taken to the laboratory and the amount of pentachlorophenol is determined using an immunochemical technique known as ELISA. Immunochemical analysis techniques are reported to be superior in selectivity and sensitivity over widely used classical methods such as GC-ECD and GC-MS (Wuske *et al.*, 1998). PSEP is a tool that can be used to discover and monitor 'hot spots' of pentachlorophenol emissions within the indoor environment but has not been tested in the ambient environment.

Other unconventional technologies that have shown some promise as future methods, such as multi-stage mass spectrometry (MS/MS) and optical remote sensing (Aragon *et al.*, 2000; Mukund *et al.*, 1995), were not researched in-depth as they are currently considered wholly unsuited for ambient monitoring of pentachlorophenol.

Table 9 **Method Advantages and Disadvantages**

Method	Advantages	Disadvantages
US EPA TO-4A	Low detection limits Effective for a broad range of pesticides/PCBs PUF is reusable Low blanks Excellent collection and retention efficiencies	Breakdown of PUF adsorbent may occur Contamination of glassware may limit detection limits Loss of some semi-volatile organics during storage Extraneous organics may interfere Difficulty in identifying individual pesticides and PCBs using some detectors
US EPA TO-10A	Easy field use Proven methodology Easy to clean Effective for a broad range of compounds Portability Good retention of compounds	Detectors are subject to responses from a variety of compounds other than target analytes PCBs, dioxins and furans may interfere Certain organochlorine pesticides can make accurate quantitation difficult May not be sensitive enough for all target analytes in ambient air
NIOSH Method 5512	NA	NA
OSHA Method 39	More convenient than NIOSH method No known interferences Rapid, sensitive and precise	Method has not been field tested Sampling tubes are not commercially available

*NA denotes not available.

6.0 AMBIENT GUIDELINES

Current and/or recommended and proposed ambient guidelines of other jurisdictions in Canada, United States and elsewhere were reviewed for pentachlorophenol. Details about guidelines that exist for each jurisdiction reviewed are presented in tabular format in this section. In general, all jurisdictions have common uses for their guidelines in practice. These uses may include:

- reviewing permit applications for sources that emit air pollutants to the atmosphere,
- investigating accidental releases or community complaints about adverse air quality for the purpose of determining follow-up or enforcement activity,
- determining whether to implement temporary emission control actions under persistent adverse air quality conditions of a short-term nature

6.1 Pentachlorophenol Air Quality Guidelines

Air quality guidelines for pentachlorophenol are summarized in Table 10. The principal approaches by which guidelines are developed include:

- Using an occupational exposure level (OEL) and dividing it by safety or adjustment factors. The most common OEL used by most state agencies is the 8-hour threshold limit value (TLV) of $500 \mu\text{g}/\text{m}^3$ for pentachlorophenol adopted by the American Conference of Governmental Industrial Hygienists (ACGIH). The safety or adjustment factors are intended to account for issues such as: differences between eight-hour exposures in the workplace and continuous 24-hour environmental exposures, increased susceptibility of some people in the general population versus the relatively healthy worker, and uncertainty in the margin of safety provided in an occupational exposure limit.
- Using non-carcinogenic risk assessment procedures. A no observed adverse effect level (NOAEL) – or lowest observed adverse effect level (LOAEL) if a NOAEL is unavailable – from a suitable animal or human study is used. It is then divided by a series of adjustment factors. The adjustment factors are intended to account for issues such as: differences between animals and humans, sensitivity of high risk individuals, use of a LOAEL instead of a NOAEL, and for extrapolation from less-than-lifetime exposures to chronic exposure.
- Using carcinogenic risk assessment procedures. Pre-existing cancer risk assessments performed by others (e.g. US EPA Integrated Risk Information System summary data) are used to establish ambient air levels based on acceptable levels of lifetime cancer risk, such as one in 100,000 (10^{-5}).

Table 10 Summary of Air Quality Guidelines for Pentachlorophenol

Agency	Guideline Title	Guideline Value [$\mu\text{g}/\text{m}^3$] Averaging Time:		
		1-hour	8-hour	24-hour annual
Ontario MOE	Ambient Air Quality Criterion (AAQC): Maximum point of impingement (POI) Guideline (30-min. averaging time): No guideline exists. No guideline exists.	60		20
US ATSDR				
US EPA				
Arizona DEQ	Arizona Ambient Air Quality Guidelines (AAQGs):	13	4	0.029 0.2 2
California EPA	Chronic reference exposure level (REL): Risk specific concentration (RsC): ¹ No guideline exists.			
Louisiana DEQ	Threshold Effects Exposure Limit (TEL): Allowable Ambient Limit (AAL):		0.01	0.01
Massachusetts DEP	Initial threshold screening level (ITSL): Initial risk screening level (IRSL):		100	0.03 0.3
Michigan DEQ	Secondary risk screening level (SRSL): 24-hour ambient air limit (AAL): Annual AAL:		1.786	1.19
New Hampshire DES				
New Jersey DEP	Risk assessment approach is used: Risk specific concentration (RsC): ¹ Acceptable ambient level (AAL): Maximum acceptable ground-level concentration (MAGLC): ²	0.025	0.003	0.29
North Carolina ENR				
Ohio EPA	Maximum acceptable ambient concentration (MAAC): No guideline exists.	11.9		
Oklahoma DEQ			5	
Rhode Island DEM				
Texas CEQ	Effects screening level (ESL): Hazardous ambient air standard (HAAS):	5		0.5 1.19 0.33
Vermont ANR	Acceptable source impact level (ASIL): Ambient air concentration (AAC): ² No guideline exists.		12	
Washington DOE				
Wisconsin DNR	No guideline exists.			
New Zealand MOE	No guideline exists.			
The Netherlands (RIVM)	No guideline exists.			
World Health Organization	No guideline exists.			

¹The RsC is not used for any specific purposes by the respective agency. It is shown here to illustrate an exposure concentration in air associated with

an inhalation unit risk or oral unit factor used by the agency and a 1 in 100,000 lifetime cancer risk (risk criteria used in Alberta); ² Proposed.

6.1.1 *Canada*

The Ontario Ministry of the Environment (MOE) adopted an Ambient Air Quality Criterion (AAQC) of $20 \mu\text{g}/\text{m}^3$ as a 24-hour guideline. Ontario MOE uses a maximum point of impingement (POI) guideline of $60 \mu\text{g}/\text{m}^3$ based on a 30-minute averaging time.

6.1.2 *United States*

The US Agency for Toxic Substances and Disease Registry (ATSDR) and the US Environmental Protection Agency (EPA) do not have air quality criteria for pentachlorophenol. Several state agencies – Arizona, Michigan, and New Jersey – use the US EPA's Integrated Risk Information System (IRIS) oral slope factor of 0.12 per $\text{mg}/\text{kg}/\text{day}$ along with a body weight of 70 kg and a breathing rate of $20 \text{ m}^3/\text{day}$ to develop air quality guidelines for annual averaging periods.

A number of states use an occupational exposure level (OEL) and divide it by safety or adjustment factors to develop their guidelines. The states of New Hampshire, Ohio, Oklahoma, Texas, Vermont, and Wisconsin adopted guidelines with averaging times ranging from 1-hour to greater than one-year based on the American Conference of Governmental Industrial Hygienists (ACGIH) 8-hour time weighted average occupational exposure limit (OEL) of $0.5 \text{ mg}/\text{m}^3$ for pentachlorophenol. The state of Arizona uses the National Institute for Occupational Safety and Health (NIOSH) relative exposure level (REL) of $0.5 \text{ mg}/\text{m}^3$ divided by safety factors for their 24-hour guideline.

6.1.3 *International Agencies*

The New Zealand Ministry of Environment and Ministry of Health, the Netherlands National Institute of Public Health, and the World Health Organization do not have air quality criteria for pentachlorophenol.

The range of air quality guidelines proposed and/or adopted for pentachlorophenol by various agencies for protection of human receptors is shown in Figure 1 for different averaging time periods.

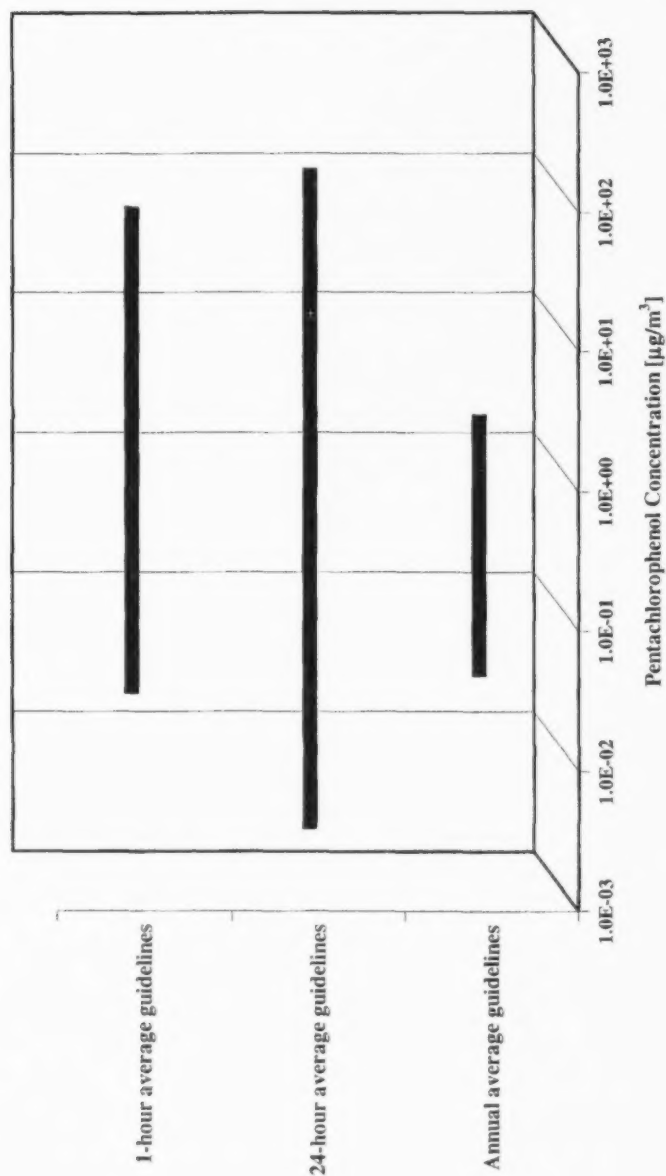


Figure 1 Range of Air Quality Guidelines for Pentachlorophenol Proposed by Various Agencies for Protection of Human Receptors

7.0 DISCUSSION

When establishing an ambient air guideline in the form of a concentration limit with a corresponding duration (i.e. averaging time), a number of factors may be taken into account for an air pollutant:

- nature of adverse health effects and conditions of exposure (e.g. exposure concentrations and duration) associated with these effects,
- estimated or actual degree of exposure of receptors, and in particular receptor groups that may be sensitive to the air pollutant,
- availability and suitability of approaches for screening and estimating ambient ground-level concentrations in order to compare to the guidelines for permit applications or other situations.

Ambient air guidelines in the form of a short-term (acute) and long-term (chronic) duration are discussed below for pentachlorophenol. Ideally the guidelines would serve to address exposures related to humans, animals and vegetation. No direct exposure-related information was obtained for vegetation; therefore the discussion emphasizes human and animal (as surrogates for human) exposures.

7.1 Acute Exposure Conditions

Pentachlorophenol has high potential toxicity, regardless of the route, length, and frequency of exposure. It is readily absorbed through cell membranes of the lungs, gastrointestinal tract and skin. Once absorbed, it is distributed to the adipose tissue, liver, lungs, kidneys, blood, brain and spleen. There is evidence for long-term accumulation and storage of pentachlorophenol, with higher concentrations detected in the liver and kidney, and lower concentrations in body fat, brain and muscle tissue.

The acute effects of pentachlorophenol reported in humans include:

- irritation of exposed epithelial tissue (eyes, nose, throat) at concentrations greater than 1 mg/m^3 (0.09 ppm) based on exposures reported as an 8-hour TWA,
- a rise in metabolic rate, hypothermia and effects on the central nervous system, respiratory system, cardiovascular system, liver and blood (concentrations not specified).

Studies on infants and children suggest a greater susceptibility of children to acute pentachlorophenol toxicity. Acute and sub-acute inhalation exposure of animals to pentachlorophenol has been reported to affect the respiratory system, blood, liver, kidney and adrenal system. Most of these effects have been observed based on exposure concentrations ranging from 3 to 21.4 mg/m^3 (0.3 to 1.9 ppm) in studies where exposures occurred four hours/day,

six days/week over four months. Comparisons of effects in acute inhalation and oral studies on rats and rabbits indicate that pentachlorophenol is significantly more toxic via inhalation than ingestion.

Most of the agencies reviewed have short-term (1-hour and/or 24-hour) air quality guidelines for pentachlorophenol for acute exposure conditions. Six state agencies – California, New Hampshire, Ohio, Oklahoma, Texas, and Wisconsin – adopted short-term guidelines for pentachlorophenol from occupational exposure limits (OELs). The use of OELs for the development of ambient guidelines is cautioned. There are limitations in the direct and indirect application of OELs for ambient air quality guidelines for a number of reasons:

- OELs are based on the information gathered in workplace, through experience from medical research and practice, from experimental human and animal studies, and from a combination of these sources. Often they are based upon averaged tolerated doses from actual repeated industrial exposures. In this respect, they would be considered very accurate at predicting human adverse health effects in industrial exposure situations.
- OELs are determined for a population of workers who are essentially healthy and who fall within a working age group of about 17 to 65 years. These individuals are supposedly in the prime of life, and potentially less susceptible to the effects of hazardous substances than other members of the public. Individuals vary in sensitivity or susceptibility to hazardous substances; with the elderly and infants in general being more susceptible than healthy workers.
- For most substances, a worker during a normal work schedule (eight hours per day, five days per week) receives 40 hours of exposure per week with daily breaks and extended weekend periods in which the body may rid itself of the accumulated substances before elevated levels are reached. For a person living continuously in an environment containing such substances, however, these recovery periods do not exist.

For these reasons, agencies using OELs have a policy of adjusting them downward with the use of safety or adjustment factors to derive guidelines for environmental (ambient) settings. The OELs are considered surrogates for benchmark values for ambient exposures only because they tend to be based upon a large body of toxicological, epidemiological, and/or clinical evidence pertaining to human exposure (albeit in the workplace). Uncertainty exists in terms of whether too much (or too little) safety is inherent in ambient air guidelines developed from OELs.

7.2 Chronic Exposure Conditions

In human occupational studies, chronic pentachlorophenol inhalation has been reported to be associated with effects on the respiratory system, endocrine system, central nervous system and reproductive system. Adverse effects on the liver and blood were also reported. However, those occupational studies carried a high potential for confounding exposure factors that limits their interpretation. Consequently, exposure concentrations corresponding to the reported effects are uncertain. The International Agency for Research on Cancer and the US Environmental Protection Agency have classified pentachlorophenol as a probable human carcinogen based on

limited evidence in human occupational exposures and sufficient evidence in animal studies through the oral pathway. No chronic inhalation animal studies were identified. Chronic animal ingestion studies reported effects on the liver and kidney; endocrine and reproductive system effects; developmental effects; and, carcinogenicity.

Most of the agencies whose air quality guidelines were reviewed have chronic (long-term) guidelines for pentachlorophenol. The states of California, Michigan, and New Jersey use a carcinogenic endpoint to derive their respective guideline – using the US EPA's oral unit risk of 0.12 per mg/kg/day. Several of the agencies also have a chronic guideline for non-carcinogenic effects – using occupational exposure limits (New Hampshire, Texas, and Vermont).

8.0 REFERENCES

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APPENDIX A

REVIEW OF AIR QUALITY GUIDELINES FOR PENTACHLOROPHENOL USED BY AGENCIES IN NORTH AMERICA AND ELSEWHERE

Agency:
Ontario Ministry of the Environment (OME).
Air Quality Guideline:
Ambient Air Quality Criterion (AAQC) = $20 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
24-hour averaging time.
Basis for Development:
Limiting effect based on health.
Date Guideline Developed:
Unknown.
How Guideline is Used in Practice:
Used by Ontario Ministry of Environment (OME) to represent human health or environmental effect-based values not expected to cause adverse effects based on continuous exposure.
Additional Comments:
AAQC is <u>not</u> used by OME to permit stationary sources that emit pentachlorophenol to the atmosphere. A "point of impingement" standard is used to for permitting situations.
Reference and Supporting Documentation:
Ontario Ministry of the Environment. 1999. Summary of Point Of Impingement Standards, Point Of Impingement Guidelines, and Ambient Air Quality Criteria (AAQC). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. November 1999. 12 pp.

Agency:
Ontario Ministry of the Environment (OME).
Air Quality Guideline:
Maximum point of impingement (POI) Guideline = $60 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
30-minute averaging time.
Basis for Development:
Limiting effect based on health.
Date Guideline Developed:
Unknown.
How Guideline is Used in Practice:
Used by OME to review permit applications for stationary sources that emit pentachlorophenol to the atmosphere.
Additional Comments:
n/a
Reference and Supporting Documentation:
Ontario Ministry of the Environment. 1999. Summary of Point Of Impingement Standards, Point Of Impingement Guidelines, and Ambient Air Quality Criteria (AAQC). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. November 1999. 12 pp.

Agency: US Agency for Toxic Substances and Disease Registry (ATSDR).
Air Quality Guideline: US ATSDR does not have an inhalation Minimal Risk Level (MRL) for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Minimal Risk Levels (MRLs) for Hazardous Substances. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. Available at: http://www.atsdr.cdc.gov/mrls.html (accessed 22 January 2003).

Agency: US Environmental Protection Agency (EPA).
Air Quality Guideline: US EPA does not have air quality criteria for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: US Environmental Protection Agency. Integrated Risk Information System. http://www.epa.gov/iris/ (accessed 22 January 2003).

Agency:

Arizona Department of Environmental Quality (DEQ)

Air Quality Guideline:

Arizona Ambient Air Quality Guidelines (AAAQG) = $13 \mu\text{g}/\text{m}^3$ (1-hour averaging time)

$4 \mu\text{g}/\text{m}^3$ (24-hour averaging time)

$0.029 \mu\text{g}/\text{m}^3$ (annual averaging time)

Averaging Time To Which Guideline Applies:

See above.

Basis for Development:

1-hour AAAQG: Unknown. However, most 1-hour AAAQGs are developed by dividing the American Conference of Governmental Industrial Hygienists (ACGIH) Short Term Exposure Limit (STEL) or other short term standard or guideline divided by 120, or the 24-hour AAAQG multiplied by 3.8. The factor for calculating a 1-hour AAAQG using a STEL represents a conversion factor that converts a 15-minute exposure into a 1-hour exposure, and a safety factor of 30 to protect the most sensitive members of the population such as children and the elderly. The factor of 3.8 – used in the calculation of a 1-hour AAAQG based upon the 24-hour AAAQG – represents the proportional difference in the lowest observed adverse effect level for 24-hour and 1-hour exposure to a common irritant (SO_2) in human subjects.

24-hour AAAQG: Developed by dividing the National Institute for Occupational Safety and Health (NIOSH) relative exposure level (REL) of $0.5 \text{ mg}/\text{m}^3$ for pentachlorophenol by 126. The factor of 126 incorporates the conversion of an 8-hour, 5-day workweek to a 24-hour, 7-day week of 4.2, and a safety factor of 30 to protect the most sensitive members of the population such as children and the elderly.

Annual average AAAQG: Developed for possible, probable and known human carcinogens. The US EPA Integrated Risk Information System (IRIS) oral slope factor of 0.12 per $\text{mg}/\text{kg}/\text{day}$ was used along with a target excess lifetime cancer risk of 1-in-one-million, a body weight of 70 kg, and a breathing rate of $20 \text{ m}^3/\text{day}$ to estimate the annual average AAAQG.

Date Guideline Developed:

Updated in May 1999.

How Guideline is Used in Practice:

AAAQGs are not intended to be used as standards. Rather, they are intended to provide health-based guidelines that may be useful in making environmental risk management decisions.

Additional Comments:

AAAQGs are residential screening values that are protective of human health, including children. AAAQGs consider human health risk from inhalation of contaminants in ambient air. They do not take into account odor thresholds or threats to wildlife. Chemical concentrations in air that exceed AAAQGs may not necessarily represent a health risk. Rather, when contaminant concentrations exceed the guidelines, further evaluation may be necessary.

Reference and Supporting Documentation:

Arizona. 1999. 1999 Update - Arizona Ambient Air Quality Guidelines (AAAQGs). Prepared by The Office of Environmental Health. Prepared for Air Programs Division, Arizona Department of Environmental Quality, Phoenix, AZ. May 11, 1999. 20 pp.

Agency:
California Environmental Protection Agency (Cal EPA)
Air Quality Guideline:
Chronic reference exposure level (REL) = $0.2 \mu\text{g}/\text{m}^3$
Risk specific concentration (RsC) corresponding to 1 in 100,000 risk = $2 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
Continuous (daily) exposure over a lifetime.
Basis for Development:
Chronic REL: Unknown. Chronic REL represents a toxicity value originally developed by California Air Pollution Control Officers Association (CAPCOA) and not indicated as being adopted by Cal EPA Office of Environmental Health Hazard Assessment (OEHHA).
RsC: Based on an inhalation unit risk factor of $5.1 \text{ E-}6$ per $\mu\text{g}/\text{m}^3$ calculated from the linearized multistage procedure using data from a study in mice.
Date Guideline Developed:
Chronic REL: 1992.
Inhalation unit risk factor: 1999.
How Guideline is Used in Practice:
Chronic RELs are for use in facility health risk assessments conducted for the AB 2588 Air Toxics "Hot Spots" Program.
The risk specific concentration (RsC) is not used for any specific purposes by Cal EPA and is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor derived by Cal EPA and a 1 in 100,000 lifetime cancer risk.
Additional Comments:
n/a
Reference and Supporting Documentation:
California Environmental Protection Agency (Cal EPA). 1999. Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Cal EPA. Oakland, CA. March 1999.
California Air Pollution Control Officers Association (CAPCOA). 1992. Air Toxics "Hot Spots" Program Risk Assessment Guidelines, Prepared by AB2588 Risk Assessment Committee of CAPCOA, Sacramento, CA. January 1992.
California Office of Environmental Health Hazard Assessment (OEHHA)/Air Resources Board (ARB). 2001. Approved Chronic Reference Exposure Levels and Target Organs. Table 3 (last updated 13 September 2001). Available at: www.arb.ca.gov/toxics/healthval/chronic.pdf (accessed 22 January 2003).

Agency:

Louisiana Department of Environmental Quality (DEQ)

Air Quality Guideline:

Louisiana DEQ does not have an air quality guideline for this chemical.

Averaging Time To Which Guideline Applies:

n/a

Basis for Development:

n/a

Date Guideline Developed:

n/a

How Guideline is Used in Practice:

n/a

Additional Comments:

n/a

Reference and Supporting Documentation:

Louisiana Administrative Code (LAC). Title 33 Environmental Quality, Part III Air, Chapter 51. Comprehensive Toxic Air Pollutant Emission Control Program. Louisiana Department of Environmental Quality. Baton Rouge, LA.

Agency: Massachusetts Department of Environmental Protection (DEP)
Air Quality Guideline: Threshold Effects Exposure Limit (TEL) = $0.01 \mu\text{g}/\text{m}^3$ (24-hour averaging time) Allowable Ambient Limit (AAL) = $0.01 \mu\text{g}/\text{m}^3$ (annual average)
Averaging Time To Which Guideline Applies: See above.
Basis for Development: Unknown.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: Information could not be obtained to identify how the guideline is used in practice, but it is expected that the guideline is used in some manner to meet state level permitting.
Additional Comments: n/a
Reference and Supporting Documentation: Massachusetts Department of Environmental Protection (DEP). 1995. Revised air guidelines [updated list of 24-hour average Threshold Effects Exposure Limit (TEL) values and annual average Allowable Ambient Limit (AAL) values]. Massachusetts DEP, Boston, MA. 6 December 1995. Memorandum available at: http://www.state.ma.us/dep/ors/files/aallist.pdf (accessed 22 January 2003).

Agency:
Michigan Department of Environmental Quality (DEQ)
Air Quality Guideline:
Initial threshold screening level (ITSL) = $100 \mu\text{g}/\text{m}^3$ (24-hour averaging time)
Initial risk screening level (IRSL) = $0.03 \mu\text{g}/\text{m}^3$ (annual average)
Secondary risk screening level (SRSL) = $0.3 \mu\text{g}/\text{m}^3$ (annual average)
Averaging Time To Which Guideline Applies:
See above.
Basis for Development:
Initial threshold screening level (ITSL): Unknown.
The IRSL and SRSL are based on the US EPA IRIS oral unit risk factor of 0.12 per mg/kg/day, body weight of 70 kg, breathing rate of $20 \text{ m}^3/\text{d}$ and target excess lifetime cancer risks of 1 in 1,000,000 and 1 in 100,000 risk, respectively.
Date Guideline Developed:
1996.
How Guideline is Used in Practice:
There are two basic requirements of Michigan air toxic rules. First, each source must apply the best available control technology for toxics (T-BACT). After the application of T-BACT, the emissions of the toxic air contaminant cannot result in a maximum ambient concentration that exceeds the applicable health based screening level for non-carcinogenic effects (ITSL). Application of an ITSL is required for any new or modified emission source or sources for which a permit to install is requested and which emits a toxic air contaminant.
Additional Comments:
The applicable air quality screening level for chemical treated as non-carcinogens by Michigan DEQ is the ITSL. There are two health based screening levels for chemical treated as carcinogens by Michigan DEQ: the initial risk screening level (IRSL) – based on an increased cancer risk of one in one million, and the secondary risk screening level (SRSL) – based on as an increased cancer risk of 1 in 100,000.
Reference and Supporting Documentation:
Michigan Administrative Code (MAC). Air Pollution Control Rules. Part 2 Air Use Approval, R 336.1201 - 336.1299. Air Quality Division, Department of Environmental Quality. Lansing, MI.

Agency: New Hampshire Department of Environmental Services (DES)
Air Quality Guideline: 24-hour ambient air limit (AAL) = $1.786 \mu\text{g}/\text{m}^3$ Annual ambient air limit (AAL) = $1.19 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies: See above.
Basis for Development: The AALs were developed in the following manner: 24-hour Ambient Air Limit – The American Conference of Governmental Industrial Hygienists (ACGIH) 8-hour time weighted average occupational exposure limit (OEL) of $0.5 \text{ mg}/\text{m}^3$ is divided by a safety factor (SF) of 100 and a time adjustment factor (TAF) of 2.8. Annual Ambient Air Limit – The American Conference of Governmental Industrial Hygienists (ACGIH) 8-hour time weighted average occupational exposure limit (OEL) of $0.5 \text{ mg}/\text{m}^3$ is divided by a safety factor (SF) of 100 and a factor of 4.2.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: AALs are used by New Hampshire DES to review permit applications for sources that emit pentachlorophenol to the atmosphere. Sources are regulated through a statewide air permitting system and include any new, modified or existing stationary source, area source or device.
Additional Comments: n/a
Reference and Supporting Documentation: New Hampshire Administrative Rule. Chapter Env-A 1400. Regulated Toxic Air Pollutants. New Hampshire Department of Environmental Services. Concord, NH.

Agency: New Jersey Department of Environmental Protection (DEP)
Air Quality Guideline: Applicants are required to carry out a risk assessment in conjunction with applying for an air pollution control pre-construction permit. In the case of pentachlorophenol, it is assumed that the US EPA IRIS oral unit risk factor of 0.12 per mg/kg/day, body weight of 70 kg, and breathing rate of 20 m ³ /d is used to calculate a lifetime cancer risk for sources that emit pentachlorophenol to the atmosphere.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: Assumed to be based on US EPA Integrated Risk Information System (IRIS) data.
Date Guideline Developed: December 1994.
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: New Jersey Administrative Code (NJAC). Title 7, Chapter 27, Subchapter 8. Permits and Certificates for Minor Facilities (and Major Facilities without an Operating Permit). New Jersey Department of Environmental Protection. Trenton, NJ. New Jersey Department of Environmental Protection. 1994. Technical Manual 1003. Guidance on Preparing a Risk Assessment for Air Contaminant Emissions. Air Quality Permitting Program, Bureau of Air Quality Evaluation, New Jersey Department of Environmental Protection. Trenton, NJ. Revised December 1994.

Agency: North Carolina Department of Environment and Natural Resources (ENR)
Air Quality Guideline: Acceptable ambient level (AAL) = $0.025 \mu\text{g}/\text{m}^3$ (1-hour averaging time) $0.003 \mu\text{g}/\text{m}^3$ (24-hour averaging time)
Averaging Time To Which Guideline Applies: See above.
Basis for Development: Not stated.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: A facility emitting pentachlorophenol must limit its emissions so that the resulting modeled ambient levels at the property boundary remain below the health-based acceptable ambient level (AAL).
Additional Comments: n/a
Reference and Supporting Documentation: North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2D.1100 – Air Pollution Control Requirements (Control of Toxic Air Pollutants). North Carolina Department of Environment and Natural Resources. Raleigh, NC. North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2Q.0700 – Air Quality Permit Procedures (Toxic Air Pollutant Procedures). North Carolina Department of Environment and Natural Resources. Raleigh, NC.

Agency:

Ohio Environmental Protection Agency (EPA)

Air Quality Guideline:

Maximum acceptable ground-level concentration (MAGLC) = $11.9 \mu\text{g}/\text{m}^3$ (proposed)

Averaging Time To Which Guideline Applies:

1-hour averaging time.

Basis for Development:

$$\text{MAGCL} = \frac{\text{TLV}}{10} \times \frac{8 \text{ hr}}{24 \text{ hr}} \times \frac{5 \text{ d}}{7 \text{ d}} = \frac{\text{TLV}}{42}$$

The TLV is the ACGIH 8-hour time weighted average occupational exposure limit (OEL) of $0.5 \text{ mg}/\text{m}^3$. The TLV is adjusted by a safety factor of 10 to take into account greater susceptibility of the general population in comparison to healthy workers. The 8/24 and the 5/7 multipliers are used to relate the exposure to longer than 40-hour time periods and ascertain that the individual's total exposure will be no greater than that allowed by the TLV.

Date Guideline Developed:

January 1994 (proposed).

How Guideline is Used in Practice:

Used by Ohio EPA to review permit applications for sources that emit pentachlorophenol to the atmosphere.

Additional Comments:

n/a

Reference and Supporting Documentation:

Ohio Environmental Protection Agency (EPA). 2003. Review of New Sources of Toxic Emissions. Air Toxics Unit, Division of Air Pollution Control, Ohio EPA. Columbus, OH. 11 pp (available at: <http://www.epa.state.oh.us/dapc/atu/atu.html>, accessed 22 January 2003).

Ohio Environmental Protection Agency (Ohio EPA). 1994. Review of New Sources of Air Toxic Emissions. Proposed for Public Comment. Division of Air Pollution Control, Ohio EPA. Columbus, OH. January 1994. 31 pp.

Agency:
Oklahoma Department of Environmental Quality (DEQ)
Air Quality Guideline:
Maximum acceptable ambient concentration (MAAC) = $5 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
24-hour averaging time.
Basis for Development:
The MAAC is the TLV for pentachlorophenol (ACGIH 8-hour time weighted average occupational exposure limit – OEL – of $0.5 \text{ mg}/\text{m}^3$) divided by a factor of 100. In this case, the factor of 100 is applied to substances that are considered by Oklahoma DEQ to be a substance of high toxicity.
Date Guideline Developed:
Not stated.
How Guideline is Used in Practice:
MAACs are used by Oklahoma DEQ to review permit applications for sources that emit pentachlorophenol to the atmosphere.
Additional Comments:
n/a
Reference and Supporting Documentation:
Oklahoma Administrative Code (OAC). Title 252. Chapter 100. Air Pollution Control. 100:252-41 - Control of Emission of Hazardous and Toxic Air Contaminants. Oklahoma Department of Environmental Quality. Oklahoma City, OK.
Oklahoma Department of Environmental Quality (DEQ). 2002. Air Toxics Partial Listing [maximum acceptable ambient concentrations (MAAC) for air toxics]. Oklahoma City, OK. Available at: http://www.deq.state.ok.us/AQDNew/toxics/listings/pollutant_query_1.html (accessed 23 January 2003).

Agency: Rhode Island Department of Environmental Management (DEM)
Air Quality Guideline: Rhode Island DEM does not have an air quality guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: Rhode Island Department of Environmental Management. 1992. Air Pollution Control Regulation No. 22. Division of Air and Hazardous Materials, Rhode Island Department of Environmental Management. Providence, RI. Amended 19 November 1992.
Agency: Texas Commission on Environmental Quality (CEQ) – formerly Texas Natural Resource Conservation Commission (TRNCC)
Air Quality Guideline: Short-term effects screening level (ESL) = $5 \mu\text{g}/\text{m}^3$ Long-term effects screening level (ESL) = $0.5 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:

1-hour averaging time for short-term ESL. Annual averaging time for long-term ESL.
Basis for Development: Short-term Effects Screening Level – The ACGIH TLV – 8-hour time weighted average occupational exposure limit (OEL) of 0.5 mg/m^3 – is divided by a safety factor of 100. Long-term Effects Screening Level – The ACGIH TLV – 8-hour time weighted average occupational exposure limit (OEL) of 0.5 mg/m^3 – is divided by a safety factor of 1000.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in air. ESLs are based on data concerning health effects, odor nuisance potential, effects with respect to vegetation, and corrosion effects. They are not ambient air standards. If predicted or measured airborne levels of a chemical do not exceed the screening level, adverse health or welfare effects would not be expected to result. If ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem, but rather, triggers a more in-depth review.
Additional Comments: n/a
Reference and Supporting Documentation: Texas Natural Resource Conservation Commission (TNRCC) 2001. Toxicology & Risk Assessment (TARA) Section Effects Screening Levels. http://www.tnrcc.state.tx.us/permitting/tox/index.html (accessed 23 January 2003).

Agency:
Vermont Agency of Natural Resources (ANR)
Air Quality Guideline:
Hazardous ambient air standard (HAAS) = $1.19 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
Annual average.
Basis for Development:
Unknown, however the HAAS is equivalent to the TLV for pentachlorophenol (ACGIH 8-hour time weighted average occupational exposure limit – OEL – of $0.5 \text{ mg}/\text{m}^3$) divided by a factor of 420. The factor of 420 is an adjustment representing a safety factor of 100 and 8/24 and 5/7 multipliers to convert 8-hour per 24-hour day and 5-day per 7-day week occupational exposures to continuous exposures.
Date Guideline Developed:
Not stated.
How Guideline is Used in Practice:
HAASs are used by Vermont ANR to review permit applications for stationary sources that emit pentachlorophenol to the atmosphere.
Additional Comments:
n/a
Reference and Supporting Documentation:
Vermont Air Pollution Control Regulations. 2001. State of Vermont Agency of Natural Resources. Air Pollution Control Division. Waterbury, VT. 29 November 2001. 187 pp.

Agency:
Washington State Department of Ecology (DOE)
Air Quality Guideline:
Acceptable source impact level (ASIL) = $0.33 \mu\text{g}/\text{m}^3$
Averaging Time To Which Guideline Applies:
Annual average.
Basis for Development:
Unknown.
Date Guideline Developed:
Unknown.
How Guideline is Used in Practice:
ASILs are used by Washington State DOE to review permit applications for sources that emit pentachlorophenol to the atmosphere.
Additional Comments:
n/a
Reference and Supporting Documentation:
Washington Administrative Code (WAC). Chapter 173-460 WAC. Controls For New Sources Of Toxic Air Pollutants. Washington State Department of Ecology. Olympia, WA.

Agency:

Wisconsin Department of Natural Resources (DNR)

Air Quality Guideline:

Ambient air concentration (AAC) = $12 \mu\text{g}/\text{m}^3$ (proposed)

Averaging Time To Which Guideline Applies:

24-hour averaging time.

Basis for Development:

Unknown. However the AAC is equivalent to the ACGIH TLV – 8-hour time weighted average occupational exposure limit (OEL) of $0.5 \text{ mg}/\text{m}^3$ – divided by a safety factor of 42 and rounded. The factor of 42 is a common adjustment representing a safety factor of 10 and 8/24 and 5/7 multipliers to convert 8-hour per 24-hour day and 5-day per 7-day week occupational exposures to continuous exposures.

Date Guideline Developed:

Not stated.

How Guideline is Used in Practice:

AACs are used by Wisconsin DNR to review permit applications for sources that emit pentachlorophenol to the atmosphere.

Additional Comments:

n/a

Reference and Supporting Documentation:

Wisconsin Administrative Code (WAC). Air Pollution Control Rules. Chapter NR 445. Control of Hazardous Pollutants. Wisconsin Department of Natural Resources. Madison WI.

Agency:
New Zealand Ministry for the Environment (MOE) and New Zealand Ministry of Health (MOH)
Air Quality Guideline:
New Zealand MOE and MOH does not have air quality criteria for this chemical.
Averaging Time To Which Guideline Applies:
n/a
Basis for Development:
n/a
Date Guideline Deveoped:
n/a
How Guideline is Used in Practice:
n/a
Additional Comments:
n/a
Reference and Supporting Documentation:
New Zealand Ministry for the Environment and Ministry of Health (New Zealand). 2000. Proposals for Revised and New Ambient Air Quality Guidelines. Discussion Document. Air Quality Technical Report No 16. Prepared by the Ministry for the Environment and the Ministry of Health. December 2000. 79 pp.

Agency:
The Netherlands National Institute of Public Health and the Environment (RIVM)
Air Quality Guideline:
RIVM does not have air quality criteria for this chemical.
Averaging Time To Which Guideline Applies:
n/a
Basis for Development:
n/a
Date Guideline Developed:
n/a
How Guideline is Used in Practice:
n/a
Additional Comments:
n/a
Reference and Supporting Documentation:
The Netherlands National Institute of Public Health and the Environment (RIVM). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. RIVN Report 711701 025. RIVN, Bilthoven, The Netherlands. March 2001. 297 pp.

Agency:
World Health Organization (WHO)
Air Quality Guideline:
WHO does not have air quality criteria for this chemical.
Averaging Time To Which Guideline Applies:
n/a
Basis for Development:
n/a
Date Guideline Developed:
n/a
How Guideline is Used in Practice:
n/a
Additional Comments:
n/a
Reference and Supporting Documentation:
World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91. WHO Regional Office for Europe, Copenhagen. 273 pp